REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME III.

Session of 1902–1903.

EDITED BY

GEORGE CARPENTER, M.D.

Hon. Secretaries:

GEORGE CARPENTER, M.D.,
THEODORE FISHER, M.D. (Bristol),
GEORGE A. SUTHERLAND, M.D.

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GEORGE CARPENTER, M.D.
THEODORE FISHER, M.D.
R. CLEMENT LUCAS, B.S.
GEORGE A. SUTHERLAND, M.D.

OFFICERS AND MEMBERS OF THE COUNCIL FOR THE SESSION OF 1903-1904.

ELECTED AT THE ANNUAL GENERAL MEETING, JULY THE 23RD, 1903.

Council (1903-1904).

J. FORD ANDERSON, M.D.
HERBERT L. CARRE-SMITH.
CHARLES W. CHAPMAN, M.D.
HENRY J. CURTIS, B.S.
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ROBERT H. PARRY (Glasgow).
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SYDNEY STEPHENSON, C.M.
HAROLD J. STILES, C.M. (Edinburgh).
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RICHARD H. A. WHITELOCKE, C.M. (Oxford).
ALFRED W. WILLS, M.D.

Honorary Treasurer.

R. CLEMENT LUCAS, B.S.

Honorary Secretaries.

GEORGE CARPENTER, M.D.
THEODORE FISHER, M.D. (Bristol).
GEORGE A. SUTHERLAND, M.D.

LIST OF OFFICE-BEARERS SINCE THE FORMATION OF THE SOCIETY IN THE YEAR 1900-1901.

Dates.	Chairmen of Council.	Honorary Secretaries.		Honorary Treasurer.	Editor of the 'Reports.'
		London.	Provincial.		
1900-1	A. E. Sansom, M.D.	Sydney Stephenson, C.M.	Charles H. Willey, C.M. (Sheffield).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1901-2	A. E. Sansom, M.D.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1902-3	A. H. Tubby, M.S.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.

RULES.

Name.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

Object of the Society.

2. The object of the Society shall be the study of disease and of allied subjects in children.

Constitution.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

Qualification for Membership.

4. All duly registered medical men shall be eligible for membership.

Officers.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council, and manage the affairs of the Society.

Ordinary Members.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

Form of Admission.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

Temporary Members.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

Honorary Members.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

Resignation and Expulsion of Members.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially

for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

Subscriptions.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

Election of Officers.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer, may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

Honorary Treasurer.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and business-like way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one of the Honorary Secretaries. He shall prepare an annual state-

ment of accounts, which shall be audited by two ordinary members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

Honorary Secretaries.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society, and, unless otherwise decided by the Council, shall act as editors of all publications authorised by the Council. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

Meetings.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the

Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

Chairman.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

Publications.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

Visitors.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

Council.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the

transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded. The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. He shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

ANNUAL REPORT OF THE COUNCIL.

Presented at the General Meeting on July the 23rd, 1903.

The Council have pleasure in reporting that the Society now includes 285 members. They deplore the loss by death of F. Manley Sims, Physician to H.R.H. the Duke of Cambridge, who died in December, 1902.

The Provincial Meeting of the Society was held on June the 20th, 1903, at the Royal Alexandra Hospital for Sick Children, Brighton, under the presidency of Dr. Wayland C. Chaffey. The meeting was attended by some fifty members and visitors. Many clinical cases were shown and eleven communications were read. A Committee, consisting of Mr. Clinton Dent, Mr. Francis Jaffrey, Dr. Dawson Williams, Mr. Sydney Stephenson, and Mr. Alfred H. Tubby, was appointed to inquire into the question of the treatment of surgical tuberculosis in urban hospitals. The Council have appointed Mr. R. Clement Lucas to deliver the Wightman Lecture in the Session of 1904.

The Council appointed one of their members, Dr. Fletcher Beach, to attend the International Medical Congress at Madrid in May, 1903, as a delegate from the Society. His report is appended.

The Council have decided that the Annual Dinner of the Society shall be held after the first Ordinary Meeting of the Society in the month of October, 1903.

The Honorary Treasurer's Balance-sheet shows the Society to be in a sound state financially.

Dr. Fletcher Beach's Report.

The International Medical Congress at Madrid, 1903.
Section of Pediatrics.

This Section met in a large room on the first floor of the Royal Library and Museum, and was very well attended. The President, Dr. Francisco Criado y Aguilar, took the chair at nine o'clock on Friday morning, April the 24th, and welcomed the members.

Six Reports were read, the titles being—

"The Therapeutic Value of the Use of Serum in Diphtheria."

"The Treatment of Club-foot."

"The Treatment of Tuberculous Joints."

"The Feeding of Infants."

"The Necessity of organising Institutions called 'Gouttes de Lait' (for distributing sterilised milk to mothers) in order to diminish the Mortality of Breast-fed Children."

"Thalassotherapeutics in Childhood."

There were upwards of eighty communications, and a large number of them had to be taken as read, for the Congress only met on five mornings from nine to twelve o'clock, and two afternoons from two to five o'clock.

An Englishman, Dr. Poynton, of London, read a paper on "A microbic agent considered as the connecting link between chorea, endocarditis, and articular rheumatism in children." The close association of chorea and rheumatism had been proved by many writers, and he and Dr. Paine had produced chorea experimentally in a rabbit by inoculating it with the diplococcus of rheumatic fever, and they had found the diplococcus in the pia mater and the endothelium of the cortical vessels. They had also found micrococci in the mitral valve, the cerebral cortex, and the pia mater in a fatal case of human chorea.

Two papers were read on the "Gouttes de Lait" insti-

tutions. Dr. Peyroux, of Paris, said that these establishments were concerned almost entirely with artificial feeding, and the results had not been satisfactory on account of the difficulties connected with supervising the sterilised milk. He thought that by distributing the sterilised milk to mothers without making inquiries as to their home circumstances, and their possible ability to suckle their children, breast-feeding had been discouraged, and these institutions should not be supported.

Dr. Deutch, of Budapest, said that women who were insufficiently fed could not suckle their children properly, and milk should be distributed, and tickets for soup kitchens given to them. If the child did not gain in weight, artificial feeding should be combined with the suckling.

The titles of some of the other papers were—

"The Treatment of Tuberculosis in Children by Meat Juice and Raw Meat."

"The Production of Surgical Anæsthesia in Children by Intra-spinal Injections of One-seventh of a Grain of Cocaine."

"The Treatment of Purulent Ophthalmia in the Newborn."

"Child Mortality in Madrid."

"The Pathology and Treatment of Pseudo-hypertrophic Paralysis."

"Coryza in Suckling Infants as an Early Sign of Hereditary Syphilis."

The following resolutions were passed.

1. The food during early infancy should be mother's milk. Natural milk is always preferable to artificial, there being at present no effective substitute to take its place. The employment of wet-nurses should be regulated, legislation for the protection of infants in this direction being urgently needed; the formation of an international league for the study of infant mortality and the means of preventing it is also necessary.

2. Serum-therapy has brought about a great improve-

ment in the treatment of various diseases, such as diphtheria, scarlatina, and typhoid fever in children. Further and more detailed studies in regard to this treatment are, however, desirable.

3. In view of the obscurity in the nomenclature, classification, and nosology of diplegias, a revision of the whole chapter of infantile diplegias is recommended.

4. The section is unanimous in the wish to see multiplied on the sea-coasts marine sanatoria in view of the prophylactic and curative effects of seaside treatment. It also recommends the foundation of school colonies, climatological stations, inland sanatoria, convalescent homes, etc., for the benefit of infants.

TREASURER'S STATEMENT, 1902-1903.

Receipts.		£	s.	d.
Brought forward from 1901–1902		200	15	10
Per Cash Book:				
By Subscriptions	£280 7 0			
By Sale of Reports	10 8 0			
		290	15	0
		£491	10	10

Payments.	£	s.	d.
Medical Society	16	16	0
G. Pulman and Sons, Printing	23	3	7
Adlard and Son	146	14	5
Bale, Sons, and Danielsson	12	5	0
S. Stephenson, Esq.	8	10	0
Mr. H. Dickinson	13	10	6
Treasurer, Petty Cash	2	10	0
Dr. George Carpenter	4	0	0
Mr. Chas. Baker	0	5	0
	227	14	6
Balance	263	16	4
	£491	10	10

THE WIGHTMAN LECTURE FUND.

July 17th, 1903 ... £20 0 0

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct.

CHAS. W. CHAPMAN, } *Auditors.*
GEORGE PERNET, }

Meetings of the Society were held during the Session of 1902–1903 *at the undermentioned places.*

1. Friday, October the 17th, 1902.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. George A. Sutherland in the Chair.

2. Friday, November the 21st, 1902.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. A. E. Sansom in the Chair.

3. Friday, December the 12th, 1902.—Special Meeting for the Discussion on Tuberculous Peritonitis. The Medical Society, Chandos St., Cavendish Square, W. Mr. A. H. Tubby in the Chair.

4. Friday, January the 16th, 1903. The Medical Society, Chandos Street, Cavendish Square, W. Dr. George E. Shuttleworth (Richmond, Surrey) in the Chair.

5. Friday, February the 20th, 1903.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. Edmund Cautley in the Chair.

6. Friday, March the 20th, 1903.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. Henry Ashby (Manchester) in the Chair.

7. Friday, April the 17th, 1903.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. Lewis Marshall (Nottingham) in the Chair.

8. Friday, May the 15th, 1903.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. Frederick Taylor in the Chair.

9. Saturday, June the 20th, 1903 (Provincial Meeting).—The Royal Alexandra Hospital for Sick Children, Brighton. Dr. Wayland C. Chaffey in the Chair.

LIST OF MEMBERS OF THE SOCIETY.

OCTOBER, 1903.

(O.M. = Original Member. T. = Treasurer. S. = Secretary. P.S. = Provincial Secretary. Ed. = Editor of the Reports. C. = Member of the Council. * = Life Member. C.C. = Chairman of Council.)

Elected

O.M. Abbott, Francis Charles, M.S., Assistant-Surgeon at St. Thomas's Hospital; Surgeon at The Evelina Hospital for Sick Children; 34, Weymouth Street, W.

O.M. Abrahams, Bertram Louis, M.B., Medical-Registrar at Westminster Hospital; 14, Welbeck Street, W.

O.M. Achard, Alexander Louis, M.D., 9, Blandford Street, Manchester Square, W.

1902 Adams, Charles E., 227, Gipsy Road, S.E.

1901 Allingham, Herbert William, Surgeon to H.M. The King; Surgeon at The Great Northern Hospital; Assistant-Surgeon at St. George's Hospital; 25, Grosvenor Street, W.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks.

1903 Anderson, J. Ford, M.D., 41, Belsize Park, N.W. (C.)

1901 Andriezen, William Lloyd, M.D., 7, Apsley Terrace, Acton.

1901 Archdale, Mervyn A., M.B., East Riding Lunatic Asylum, Beverley.

Elected

1902 Armour, Donald John, M.B., Assistant-Surgeon at The West-London Hospital; Senior Assistant-Surgeon at The Belgrave Hospital for Children; 89, Harley Street, W.

O.M. Armstrong, Henry George, Medical-Officer at Wellington College; Wellington College, Berks. (C. 1900—1901.)

1901 Armstrong, John T., Selby House, Beaumont Terrace, West Kensington, W.

O.M. Ashby, Henry, M.D., Physician at The Manchester General Hospital for Children; Lecturer on Diseases of Children at Owens College; 13, St. John Street, Manchester. (C. 1900—1903.)

O.M. Barnardo, Thomas John, 18, Stepney Causeway, E.

1900 Barr, James, M.D., Physician at The Royal Infirmary, Liverpool; 72, Rodney Street, Liverpool.

1902 Barrett, Alfred Keppel, 2, Peel Place, London, W.

1901 Battams, J. Scott, 123, Lavender Hill, S.W.

O.M. Beach, Fletcher, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Winchester House, Kingston Hill, Surrey. (C. 1900—1903.)

1901 Beddard, Arthur Philip, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1903 Belfrage, Sydney H., M.D., 1, Montagu Place, W.

1901 Benjafield, W. B., M.B., "Bletchenden," Lower Edmonton, N.

1900 Blagg, Arthur F., M.D., Physician at The Orthopædic Hospital for Children, Bristol; 28, Caledonia Place, Clifton, Bristol.

1902 Blaikie, James Brunton, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 22, Grosvenor Street, W.

O.M. Bluck, Walter Cardy, M.B., Byford House, York Road, Southend-on-Sea.

O.M. Blumfeld, Joseph, M.D., Assistant-Anæsthetist at St. George's Hospital; 7, Cavendish Place, W.

1900 Bolus, Harry Boulcott, M.B., "Haddon," Beckenham, Kent.

Elected

O.M. Boyd-Wallis, Albert W., Brentwood, Essex.

1903 Braithwaite, John, M.D., Hardwick Cliff, Buxton.

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.B., Hoylake, Cheshire.

1901 Bryant, John Henry, M.D., Assistant-Physician at Guy's Hospital; 8, Mansfield Street, W.

1901 Burgess, William Milner, 43, Nicoll Road, Harlesden, N.W.

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at Paddington-Green Children's Hospital; 86, Harley Street, W.

1901 Burt, Albert Hamilton, 34, Montpelier Road, Brighton.

O.M. Burton, Charles George, 77, Church Street, Lower Edmonton, N.

1902 Buzzard, E. Farquhar, M.B., 33, Harley Street, W.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

1902 Campbell, Henry Johnstone, M.D., Senior Physician at The Bradford Royal Infirmary.

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children; 63, Great Victoria Street, Belfast.

1901 Carless, Albert, Surgeon at King's College Hospital; 10, Welbeck Street, W.

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children; 22, Northumberland Street, Edinburgh. (C. 1900—1902.)

O.M. Carpenter, George, M.D., late Physician at The Evelina Hospital for Sick Children; Assistant-Physician at the North-Eastern Hospital for Children; 12, Welbeck Street, W. (C. 1900—1901.) (S.) (Ed. 1900—1903.)

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C.)

1900 Cathcart, George C., M.B., Senior Assistant-Surgeon at The London Throat Hospital, 35, Harley Street, W.

Elected

O.M. Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children; Assistant-Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C. 1900—1903.) (*)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C. 1900—1903.)

1901 Chaldecott, John Henry, Anæsthetist at St. John's Hospital for Diseases of the Skin, at The Metropolitan Throat, Nose, and Ear Hospital, and at The Italian Hospital; 41, Welbeck Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W. (C.)

1901 Cheyne, William Watson, C.B., C.M., F.R.S., Surgeon at King's College Hospital, and at Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901—1902.)

O.M. Chubb, William L., M.D., Darenth House, Sandgate.

1901 Clapham, E. W., 48, Harley Street, W.

O.M. Clarke, Ernest, M.D., Surgeon at The Central London Ophthalmic Hospital; Ophthalmic-Surgeon at The Miller Hospital, Greenwich; 3, Chandos Street, Cavendish Square, W.

O.M. Clarke, James Jackson, M.B., Surgeon to Out-patients at The City Orthopædic and at The North-West-London Hospitals; 18, Portland Place, W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collie, Robert John, M.D., Medical-Examiner for The School Board for London; 25, Porchester Terrace, Hyde Park, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 35, Westbourne Terrace, W.

Elected

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West-London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1901 Cooper, Percy Robert, M.D, B.Sc. "Glenthorne," The Downs, Bowdon, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1901 Cox, Joshua J., M.D., Physician for Diseases of Children to The Clinical Hospital, Manchester; 38, Deansgate, Manchester.

1902 Cross, Ernest, W., "The Limes," Wallwood Park, Leytonstone.

1901 Crossley, Leonard, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 Crowley, Ralph H., M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

1902 Curtis, Henry J., B.S., Assistant-Surgeon at The Royal Hospital for Women and Children; Surgeon at the North-Eastern Hospital for Children; 152, Harley Street, W. (C.)

1901 Dawnay, Archibald Hugh Payan, Anæsthetist at The Royal Orthopædic Hospital; 48, St. Mary Abbott's Terrace, Kensington, W.

O.M. Deas, Frank, Thorndale, Merton Park, S.W.

O.M. Dent, Clinton T., M.C., Surgeon at St. George's Hospital and at The Belgrave Hospital for Children; 61, Brook Street, W. (C.)

1901 Dixon, R. Halstead, M.B., 3, Florence Terrace, Ealing Common, W.

O.M. Dockrell, Morgan, M.D., Physician at St. John's Hospital for Diseases of the Skin; 9, Cavendish Square, W.

1901 Dodd, Arthur Herbert, 49, Church Road, Hove, Sussex.

1901 Douglas, Arch. R., Royal Albert Asylum, Lancaster.

1901 Drake-Brockman, E. F., Lieutenant-Colonel I.M.S., late Professor of Physiology and Diseases of the Eye at The Medical College, Madras, and late Superintendent at The Eye Infirmary, Madras, India; 14, Welbeck Street, W.

Elected

O.M. Drew, Douglas, B.S., Surgeon at The North-Eastern Hospital for Children and at The Hospital for Women, Soho Square; 49, Devonshire Street, W.

1901 Dun, Robert Craig, M.B., Surgeon at The Liverpool Infirmary for Children; 6, Rodney Street, Liverpool.

1901 Dunlop, James, Aveley, Purfleet, Essex.

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West Derby Road, Liverpool.

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

O.M. Edmunds, Walter, M.C., Surgeon to Out-patients at The Evelina Hospital for Sick Children; Surgeon at The Tottenham Hospital; 2, Devonshire Place, W. (C. 1900—1903.)

1901 Ellis, W. McDonogh, M.D., Assistant-Physician at The Hospital for Women, Bath; Cedar Lodge, Bath.

1901 Evans, J. Howell, M.B., 28, Davies Street, Berkeley Square, W.

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W.

O.M. Fenwick, William Soltau, M.D., Senior Physician at The London Temperance Hospital, and Physician at The Evelina Hospital for Sick Children; 29, Harley Street, W.

1901 Fisher, H. Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., Pathologist at The Bristol Royal Infirmary, and Physician to Out-patients at The Bristol Hospital for Sick Children; Harley Lodge, Clifton Down, Bristol. (C. 1900—1901.) (P.S.)

1902 Flower, George John William, "Hendford," Yeovil, Somerset.

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

1903 Fortescue-Brickdale, John Matthew, M.A., M.D. Oxon., B.Ch., Registrar and Pathologist to The Royal Hospital for Children and Women, Bristol; 52, Pembroke Road, Clifton, Bristol.

Elected

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at Paddington-Green Hospital for Children; 14, Harley Street, W.

1901 Francis, J. E., Charlcote, Northam, North Devon.

1901 Freeland, Ernest Harding, Surgeon at St. George's and St. James's General Dispensary; 145, Harley Street, W.

1901 Frost, Edmund, M.B., "Chesterfield," Chesterfield Road, Eastbourne. (*)

O.M. Fuller, Andrew, Local Government Board Inspector; "The Bushes," Chase Road, Oatlands Park, Weybridge, Surrey.

1901 George, Alfred Walter, M.D., Surrey House, Shoot-up Hill, N.W.

1900 Gervis, F. Harcourt, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

O.M. Giffard, Henry Edward, Denham House, Egham.

O.M. Giles, Leonard Thomason, 13, Valley Bridge Parade, Scarborough.

1902 Goodall, Edward W., M.D., Medical Superintendent at The Eastern Hospital, Homerton, N.E.; The Eastern Hospital, N.E.

1901 Grant, J. Dundas, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. Gray, Alexander C. E., M.D., 1, Elm Park Gardens, South Kensington, W.

1901 Gullan, Archibald Gordon, M.D., 5, Canning Street, Liverpool.

O.M. Guthrie, Leonard George, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Assistant-Physician at The North-West-London Hospital; Senior Physician at The Paddington-Green Children's Hospital; 15, Upper Berkeley Street, W. (C. 1900—1903.)

O.M. Gwynne, Charles Nelson, M.Ch., Surgeon at The Children's Hospital, Sheffield; Highfield House, Sheffield. (C.)

Elected

O.M. Hawken, Giles Lockwood Lang, Surgeon at St. John's College, Hurstpierpoint, Sussex.

1901 Hawthorne, Charles Oliver, M.D., Physician at The Central London Ophthalmic Hospital; Assistant-Physician at The Royal Hospital for Children and Women; 28, Weymouth Street, W.

1901 Hayes, Thomas C., M.D., Professor of Obstetric-Medicine and Diseases of Women at King's College Hospital; 17, Clarges Street, Mayfair, W.

1901 Heywood, Charles Christopher, M.B., Winter's Buildings, St. Ann Street, Manchester.

1902 Hinds, Frank, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing.

1901 Hobhouse, Edmund, M.D., Assistant-Physician at The Sussex County Hospital; Physician at The Royal Alexandra Hospital for Children, Brighton; 36, Brunswick Place, Hove. (C.)

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C.)

O.M. Hutton, David Mount, M.D., 75, Hoghton Street, Southport.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester. (C.)

1901 Huxley, Henry, 39, Leinster Gardens, Hyde Park, W.

O.M. Jaffrey, Francis, Assistant-Surgeon at St. George's Hospital; Surgeon to Out-patients at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

Elected

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

1901 Jones, E. White, C.M., Surgeon at The Longton Hospital; Chaplin House, Longton, Staffordshire.

O.M. Jones, Robert, Surgeon at The Royal Southern Hospital, Liverpool; 11, Nelson Street, Liverpool. (C. 1900—1902.)

1903 Jones, W. Llewelyn, M.D., 58, Thomas Street, Merthyr Tydfil, South Wales.

1902 Juckes, Frank Ambrose, M.B., 14, Carfax, Horsham, Sussex.

1901 Kapadia, S. A., M.D., 49, Longridge Road, South Kensington.

1902 Kelson, William Hy., M.D., Assistant-Surgeon at The London Throat Hospital; 15, Old Burlington Street, W.

1901 Kirk, T. Sinclair, B.Ch., Surgeon at The Belfast Children's Hospital, and Assistant-Surgeon at The Royal Hospital, Belfast; 19, Great Victoria Street, Belfast. (C. 1902—1903.)

O.M. Knapton, George, Yew Bank, Bowdon, Cheshire.

O.M. Lack, Harry Lambert, M.D., Ear and Throat-Surgeon at The Children's Hospital, Paddington-Green; Surgeon at The Throat Hospital, Golden Square; 48, Harley Street, W.

1901 Lacy, Alexander Gairdner, "Saumarez," Sunninghill, Ascot, Berks.

1901 Lake, Richard, Surgeon at The Royal Ear Hospital; 19, Harley Street, W.

O.M. Lambert, Sydney H. A., M.B., Gothic House, Harrow-on-the-Hill.

1901 Langdon-Down, Percival L., M.B., 47, Welbeck Street, W.

1900 Langdon-Down, Reginald Langdon, M.B., 47, Welbeck Street, W.

O.M. Lankester, Alfred Owen, 5, Upper Wimpole Street, W.

O.M. Lawson, Arnold, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital, and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green; 12, Harley Street, W.

Elected

O.M. Lee, Philip George, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork.

1901 Leslie, W. Murray, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1901 Levy, Alfred Goodman, M.D., Physician at The Farringdon General Dispensary; 41, Devonshire Street, W.

O.M. Lewis, Percy G., M.D., Folkestone, Kent.

O.M. Lister, Thomas David, M.D., Registrar and Pathologist at The East-London Hospital for Children; 95, Wimpole Street, W.

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

O.M. Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T.) (*)

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 78, Dublin Road, Belfast. (C. 1901—1902.)

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C. 1902—1903.)

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C.)

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling.

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

Elected

1901 Makins, George Henry, C.B., Surgeon at St. Thomas's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 47, Charles Street, Berkeley Square, W.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C. 1900—1903.)

1900 May, W. Page, M.D., 9, Manchester Square, W.

1902 Maynard, John Seymour, M.B., 4, Charles Street, St. James's Square, W.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull.

1902 Miller, Charles Hewitt, The London Hospital, E.

1902 Milligan, Wyndham Anstruther, C.M., Anæsthetist at The Great Northern Central Hospital; 104, Bethune Road, N.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Assistant-Surgeon at The Children's Infirmary, Liverpool; 11, Rodney Street, Liverpool.

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester; 15, St. John Street, Manchester.

1901 Morgan, Cecil Augustus, M.B., 6, Somerset Street, Portman Square, W.

1902 Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.

1901 Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W. (C.)

1901 Morton, Samuel Ernest, 72, Burngreave Road, Sheffield.

O.M. Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford.

Elected

1902 Mummery, John Percy Lockhart, M.B., 10, Cavendish Place, W.

1901 Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.

1901 Murtz, Anton Peter, Clareville, 48, Queen's Road, Finsbury Park, N.

1901 Nabarro, David N., M.D., Pathologist at The Evelina Hospital for Sick Children; 6, Crown Office Row, Inner Temple, E.C.

1901 Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.

1902 Newbolt, George P., Surgeon at The Royal Southern Hospital, Liverpool; 42, Catherine Street, Liverpool.

1901 Nisbet, J. Tawse, M.D., 7, Croxteth Road, Liverpool.

1901 Niven, Robert, Bethnal House Asylum, Cambridge Road, N.E.

1902 Northcote, Percy, M.B., 101, Jermyn Street, S.W.

1901 Oldmeadow, Lloyd J. H., M.D., 15, Half Moon Street, Piccadilly, W.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1901 Palmer, Frederick S., M.D., 7, Gordon Place, Gordon Square, W.C.

1901 Pare, John William, C.M., 64, Brook Street, W.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital and Physician at The North-Eastern Hospital for Children; 57, Wimpole Street, W.

1902 Parkinson, Thomas W., M.D., 77, Sloane Street, S.W.

1903 Parry, Robert H., Surgeon at The Victoria Infirmary and at The Royal Hospital for Sick Children, Glasgow; 3, Royal Crescent, Queen's Park, Glasgow. (C.)

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1903 Pearson, S. Vere, M.B., Assistant-Physician at The East London Hospital for Children, Shadwell; 8, Serjeant's Inn, E.C.

Elected

1901 Pedley, R. Denison, Dental-Surgeon at The Evelina Hospital for Sick Children, S.E.; 17, Railway Approach, London Bridge, S.E.

1901 Pendlebury, Herbert S., B.C., Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. Penrose, Frank, M.D., Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; 84, Wimpole Street, W. (*)

1901 Pernet, George, Assistant at the Skin Department, University College Hospital; 77, Upper Gloucester Place, N.W. (C.)

O.M. Peskett, A. W. Chalmers, M.B., Medical-Officer at The Nottingham Children's Hospital; 15, The Ropewalk, Nottingham.

1902 Phillips, Hugh, M.B., 28, Palace Court, W.

1901 Poland, John, Senior Surgeon at The Miller Hospital, Greenwich; Surgeon at The City Orthopædic Hospital; 2, Mansfield Street, W.

1902 Potter, Bernard Elwell, M.B., Resident Medical Officer at St. George's Hanover Square Dispensary; 16, Little Grosvenor Street, W.

1901 Potts, William A., M.D., 160, Hagley Road, Birmingham.

O.M. Power, d'Arcy, M.B., Assistant-Surgeon at St. Bartholomew's Hospital and Surgeon at The Victoria Hospital for Children, Chelsea; 10A, Chandos Street, Cavendish Square, W. (C. 1900—1901.)

O.M. Prangley, Henry J., Tudor House, Anerley, S.E. (*)

1901 Pratt, Eldon, M.D., Henfield, Sussex.

1901 Pritchard, George Eric Campbell, M.B., 14, Cromwell Place, South Kensington, S.W.

1901 Quennell, Arthur, Brentwood, Essex.

1901 Raw, Nathan, M.D., Mill Road, Liverpool.

1901 Ray, John Howson, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 22, St. John Street, Manchester.

1901 Reeves, Henry A., Surgeon at The Hospital for Women and at The Royal Orthopædic Hospital; 70A, Grosvenor Street, W.

Elected

1901 Rendel, Arthur Bowen, M.B., 43, Albion Street, Hyde Park, W.

1901 Rhodes, John Milson, M.D., Didsbury.

1903 Ridley, J. Brooke, M.D., 32, Sydenham Road, Croydon.

O.M. Roberts, David Lloyd, M.D., Consulting-Obstetric-Physician at The Manchester Royal Infirmary; 11, St. John Street, Manchester.

1901 Roberts, George A., Oaklands, Langton, Tunbridge Wells.

O.M. Robinson, Henry Betham, M.S., Assistant-Surgeon at St. Thomas's Hospital and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C.)

1902 Romer, Harry, M.B., 132, Marina, St. Leonard's-on-Sea.

1901 Rose, Samuel Frank, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The North-Eastern Hospital for Children; 59, Queen Anne Street, W.

O.M. Roth, Bernard, Orthopædic-Surgeon at The Royal Alexandra Hospital for Children, Brighton: 38, Harley Street, W.

1901 Rowland, Penry W., M.B., Royal Sea-bathing Hospital, Margate.

1902 Rowlands, Robert Pugh, The College, Guy's Hospital.

1902 Roxburgh, Alex. Bruce, M.B., Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square, W. (*)

O.M. Rugg, George L., 106, Knight's Hill Road, West Norwood, S.E.

1901 Rumboll, Sydney, Cavendish House, Leeds.

O.M. Sansom, A. Ernest, M.D., Consulting-Physician at The London Hospital, and Consulting-Physician at The North-Eastern Hospital for Children; 84, Harley Street, W. (C.C. 1900—1902.) (C. 1902—1903.) (*)

O.M. Sansom, Henry Arthur, M.D., 127, West End Lane, N.W.

Elected

1901 Schilling, George, 58, Crystal Palace Park Road, Sydenham, S.E.

O.M. Sequeira, James H., M.D., Physician in Charge of the Skin Department, The London Hospital; 13, Welbeck Street, W.

1901 Shackleton, William Webb, M.B., Grove House, Bushey, Herts.

1901 Shenton, Edw. W. H., 20A, Hanover Street, Hanover Square, W.

O.M. Shuttleworth, George E., M.D., Ancaster House, Richmond Hill, Surrey. (C. 1900—1903.)

1901 Simson, Henry J. Forbes, 3, Charles Street, Berkeley Square, W.

1901 Skelding, Henry, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford.

1901 Smith, Frederick John, M.D., Senior Assistant-Physician at The London Hospital; 138, Harley Street, W. (*)

1901 Smith, Guy Bellingham, B.S., Surgeon at The Surrey Dispensary; 21, St. Thomas's Street, S.E.

1901 Smith, T. Gilbart, M.D., Physician at The London Hospital; 68, Harley Street, W.

1901 Smith, Travers, R.M., M.B., 13, Kilburn Park Road, N.W. (*)

1901 Smyth, Sidney B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast. (C.)

O.M. Snape, Ernest, M.D., 41, Welbeck Street, W.

1901 Snell, Simeon, Ophthalmic-Surgeon at The Sheffield General Infirmary; 70, Hanover Street, Sheffield.

1901 Soper, William, 307, Clapham Road.

O.M. Spokes, P. Sidney, Dental-Surgeon at University College Hospital; 4, Portland Place, W.

1903 Spriggs, Edmund Ivens, M.D., Physician to Out-patients at The Victoria Hospital for Children and at The City of London Chest Hospital; 24, St. Thomas's Street, S.E.

O.M. Staunton, Michael Cornelius, M.D., Visiting-Surgeon at The Children's Hospital, Dublin; 22, North Frederick Street, Dublin.

Elected

1901 Staveley, William H. C., 13, South Eaton Place, S.W.

O.M. Stephenson, Sydney, C.M., Ophthalmic-Surgeon at The Evelina and The North-Eastern Hospitals for Children; 33, Welbeck Street, W. (S.) (C. 1901—1903.)

1901 Steward, Francis James, M.S., Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 24, St. Thomas's Street, S.E.

O.M. Stewart, Hugh M., M.D., Walton House, Lordship Lane, S.E.

O.M. Stiles, Harold J., C.M., Surgeon at The Royal Edinburgh Hospital for Sick Children; 8, St. Colme Street, Edinburgh. (C. 1900—1901.) (C.)

1900 Street, Alfred Francis, M.D., "Burghfield," St. Mildred's Road, Westgate-on-Sea.

O.M. Summerhill, Thomas Henry, Surgeon at The Alexandra Children's Hospital, Rhyl; "Bodfor," Rhyl, Flintshire.

O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; 11, Cavendish Place, W.

O.M. Sutherland, George A., M.D., Physician at The Paddington-Green Children's Hospital; Assistant-Physician at The North-West-London Hospital; 73, Wimpole Street, W. (S.) (C. 1900—1903.)

1901 Swan, Russell H. J., M.B., 8, St. Thomas's Street, London Bridge, S.E.

1901 Swanton, James Hutchinson, M.D., 40, Harley Street, W.

1901 Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 6, St. Thomas's Street, S.E.

1901 Taylor, Frederick, M.D., Senior Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901—1902.) (C.)

O.M. Taylor, James, M.D., Physician at The National Hospital for Paralysed and Epileptic, Queen Square, and Physician at The North-Eastern Hospital for Children; 49, Welbeck Street, W. (C.)

O.M. Thomson, StClair, M.D., Physician at The Throat Hospital, Golden Square, and Surgeon at The Royal Ear Hospital; 28, Queen Anne Street, W.

Elected

1902 Thorne, Atwood, M.B., Assistant-Surgeon at The London Throat Hospital; 10, Nottingham Place, W.

1901 Thorne, W. Bezly, M.D., 53, Upper Brook Street, W.

1902 Tod, Hunter, M.B., 7, Wimpole Street, W.

1901 Trout, J. H. Ensor, 412, Monument Road, Birmingham.

O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Senior Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 25, Weymouth Street, W. (C. 1900—1902.) (C.C. 1902—1903.)

1903 Tullis, George Augustus, M.D., Holmbush, Grove Road, Southsea.

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Grove, W.

1901 Turner, H. Stanley, 16, Oakley Square, S.W.

1902 Turton, James, "Hatherley," Preston Park, Brighton.

O.M. Vacher, Francis, Consulting-Surgeon at The Wirrell Hospital for Children; "Glenmore," Shrewsbury Road, Birkenhead.

O.M. Vickery, William H., Surgeon at The Children's Hospital, Newcastle-on-Tyne; 52, Elswick Road, Newcastle-on-Tyne. (C. 1900—1901.)

1902 Walker, J. W. Thomson, C.M., 8, Cavendish Place, W. (C.)

1901 Wallace, Joseph B., M.B., 39, Battersea Rise, S.W.

1901 Walsh, David, M.D., Physician at The Western Skin Hospital; 70a, Grosvenor Street, W.

1901 Waring, John Alfred, M.B., 21, Derby Road, Nottingham.

1901 Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W. (*)

1901 Wellby, Stanley, M.B., 27, St. Julian's Farm Road, West Norwood.

1902 Westmacott, Frederick H., Aural-Surgeon; 8, St. John Street, Manchester.

Elected

1901 Whipham, Thomas Roland C., M.B., Medical-Registrar at St. George's Hospital, Physician to Out-patients at the Evelina Hospital for Children; 11, Grosvenor Street, W.

1901 Whitelocke, Richard H. A., C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C.)

O.M. Wilks, Joseph Henry, B.C., Surgeon at The Children's Hospital, Sheffield; "Fairfield," Sharrow, Sheffield.

O.M. Williams, Dawson, M.D., Physician at The East-London Hospital for Children; 2, Wyndham Place, Bryanston Square, W. (C. 1900—1902.) (*)

O.M. Williams, Edward Cecil, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 9, Whatly Road, Clifton, Bristol.

O.M. Willey, Charles H., C.M., Surgeon at The Sheffield Children's Hospital; Wostenholm Road, Sharrow, Sheffield. (P.S. 1900—1901.) (C. 1901—1902.)

1901 Wills, William Alfred, M.D., Assistant-Physician at Westminster Hospital; Senior Physician at The North-Eastern Hospital for Children; 29, Lower Seymour Street, W. (C.)

O.M. Wilson, Kemlo R. M., M.D., Hanwell, W.

1902 Wood, Edward, Glebe Lodge, Windmill Hill, Enfield, Middlesex.

1901 Woods, John Francis, M.D., Medical-Superintendent at The Hoxton House Asylum, N.; 29, Queen Anne Street, W.

1901 Wright, Douglas, 229, Camden Road, N.

1902 Wright, George A., M.B., Surgeon at The Manchester Royal Infirmary; Surgeon at The Children's Hospital, Pendlebury, Manchester; 16, St. John Street, Manchester.

**** *It is requested that any inaccuracy in the foregoing list may be notified to either of the London Honorary Secretaries.*

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REPORTS

OF

THE SOCIETY

FOR THE

STUDY OF DISEASE IN CHILDREN.

SESSION of 1902–3.

1. A CASE OF CAPILLARY ANGIOMA OF THE NECK.

By WILLIAM MILNER BURGESS.

Ronald C—, aged 4 years and 7 months, a twin, the elder and the larger, was born the 22nd of March, 1898, after an easy labour.

On April the 10th a small sessile capillary nævus was noticed on the left side of the neck, which grew rapidly. There was no history of angiomata in the parents or three other children. When he was two months old, fuming nitric acid was applied to it by means of vaccine tubes.

On June the 11th chloroform was given and galvano-puncture applied. On July the 5th chloroform was given for the second time and the actual cautery applied, the instrument being a cobbler's awl heated in a spirit-lamp. On July the 20th there was much inflammation resulting from the cautery.

On August the 19th chloroform was given for the third

and last operation—namely, puncture with the actual cautery. On August the 29th there was ulceration, which was dressed antiseptically. By September the 5th scar tissue the shape of the sole of a boot had formed, which presented some red points at the margin. On September the 28th one or two pin-points of capillary nævus tissue were touched with fuming nitric acid.

I exhibit this case as an example of puncture by the actual cautery. In my opinion, the nævus was too large for my usual practice of excision, and I also should have had to tie the external jugular vein.

It was my intention to show a similar case treated in the same way, in a young girl, but she is at a boarding school.

During over twenty years' experience of private practice among the middle classes, I have not met with many nævi, but when I have seen them they have often been multiple and mostly rapidly growing, sometimes disappearing, or stationary.

I have had one case of pulsating nævus of the anterior temporal artery, but this was of traumatic origin.

(*Exhibited October the 17th*, 1902.)

Discussion.

Mr. Francis Jaffrey said he preferred excision in all cases. He had tried the application of nitric acid, but, unless the skin were destroyed very thoroughly, the growth returned. Only in the case of nævi around the orifices, such as the anus, the vulva, and the mouth, had he applied the actual cautery. He preferred excision wherever possible.

Mr. Walter Edmunds said he had treated nævi on the face with the electric point, which was a very satisfactory way if an anæsthetic be given, so that the treatment can be well done each time. The present case had certainly been very successful under Mr. Burgess' treatment.

Dr. C. W. Chapman said that many years ago he had a case of nævus in the right eyelid, which was treated with a crochet needle which had been heated in a spirit-lamp, and the result was good.

Mr. Albert Carless congratulated Mr. Burgess on the result. Personally, he preferred to treat such cases by excision, the

results of which he had found to be good. It was most important that nævi which were tending to grow should be dealt with efficiently and radically. Moreover, an untreated nævus perhaps remained quiescent for many years, and then sometimes suddenly took on active growth. It seemed as though there was some instability of the cells of a nævus, which caused them, perhaps 30 or 40 years later, to develop, leading to growths which were almost angiosarcomatous. Some years ago, he saw a man who had been the subject of a capillary nævus of the face since birth. Suddenly it developed into a protruding angiomatous mass, something like a turkey's wattle, which began to bleed. He was taken into hospital, and operative measures were proposed, but he refused treatment.

2. A CASE OF HYPERTROPHIC STENOSIS OF THE PYLORUS WITH DILATATION OF THE STOMACH: PYLOROPLASTY: RESULT.

By LEONARD GUTHRIE, M.D.

A MALE infant, aged 4 months, was admitted into the Children's Hospital, Paddington Green, suffering from symptoms of pyloric obstruction, on August the 25th, 1902. Dr. Woodhouse, of Esher, kindly sent the following particulars.

Since six weeks of age there had been frequent vomiting, and obstinate constipation only relieved by glycerine enemata. The motions sometimes contained blood and slime. The child had occasional attacks of very severe colic accompanied by abdominal distension, and visible peristalsis, which became so marked on August the 20th as to suggest intussusception.

The vomiting persisted in spite of treatment. The child was breast-fed. The family history was unimportant.

On admission he weighed 11 lbs. The abdomen was distended and tympanitic, the coils of intestines standing out markedly. A small umbilical and a large left inguinal hernia were present. During a rectal examination a

normal motion was passed, and later in the day another motion containing mucus was evacuated after an enema. He vomited three times on the day of admission, and cried out much from abdominal pain.

For the next few days the child was very restless and uneasy, but appeared to be relieved by eructation of large quantities of flatus. The bowels acted normally, and vomiting occurred once daily.

On gently stroking the abdomen the outlines of the stomach stood out prominently, and large peristaltic waves passed from left to right across the epigastrium. The stomach became "hour-glass" in form, from bulging on either side of the recti muscles. The organ was obviously dilated, and on passing a tube for the purpose of washing it out the curve of the tube could be felt two inches below the umbilicus, whilst its end projected as far down as the right iliac fossa. Twelve to fifteen ounces of fluid could be introduced into the stomach without inducing vomiting, but a larger quantity was immediately returned.

Lavage gave considerable relief from vomiting and flatulency, and the active peristaltic movements subsided.

Great difficulty was experienced in feeding the infant; milk was undigested and produced loose, watery, and offensive motions. A diet of cream and whey seemed to suit it best. It gained half a pound a week for a fortnight, but then began to lose weight steadily. It suffered considerably from flatulence and colic, only relieved temporarily by lavage. The stomach, however, would not now hold more than twelve fluid ounces without causing emesis, and the dilatation was therefore less than it had been. But, no other improvement was manifest.

The pyloric obstruction in this case was but partial, and not so extreme as that met with in other cases; but as the infant was obviously going downhill, in spite of careful dieting and medicinal treatment, operative measures seemed to be indicated.

The opinion of the Society was sought as to the form of operation most suitable in such cases.

As regards the cause of pyloric obstruction in this disease, he inclined to the view held by Dr. John Thomson, namely, that there was spasm of the sphincter, or inco-ordination between the contraction of the sphincter and the propulsive efforts of the stomach walls.

(Exhibited October the 17th, 1902.)

Discussion.

Dr. Edmund Cautley thought the case was very difficult from the point of view of diagnosis. Possibly the dilatation of the stomach accounted for the variation in weight of the child. The cause of the dilatation seemed doubtful. It might be some pyloric obstruction, and pyloric obstruction in babies was usually due to congenital hypertrophic stenosis. There were cases of pyloric obstruction without any hypertrophy, where there was simply atresia of the pylorus. Possibly the diagnosis was dilatation secondary to congenital hypertrophic stenosis of the pylorus with incomplete obstruction. The child was four months old, and it was unusual for cases of simple hypertrophic stenosis to live as long as that. If they did, they were extremely marasmic. The spasm theory was incapable of proof. In hypertrophic stenosis of the pylorus there is marked hypertrophy of the circular muscular fibres. There seemed no reason why there should not be spasm without hypertrophy. A small amount of spasm would cause pyloric obstruction by preventing dilatation of the pylorus. One did not find hypertrophy of the muscles of the stomach in such cases, but there is an enormous hypertrophy of the circular muscular fibres of the pylorus. Again, why should a child have inco-ordination of the muscles of the pylorus? There seemed no reason why spasm of that nature should be started *in utero*, but it must have started *in utero* in some cases because the symptoms started two or three days after birth. His view was that it was a simple hyperplasia, and that, as a result of contraction of the muscular fibres, the mucous membrane got thrown into folds, and the combination of those two factors caused complete obstruction. It would be interesting if Dr. Guthrie could bring up the child without operation. He thought, as more of the cases were seen, a definite connection might be traced between the hypertrophic stenosis of infancy and that of older children, and even adults. He had encountered seven cases, and the oldest of those was fourteen weeks at the time of death. With regard to treatment, he would try a little longer the effect of washing out the stomach, and careful dieting, giving only very small quantities of equal parts of peptonised milk and water every two hours. If, in spite of treatment, the child still lost ground, an incision might be made.

The views of surgeons about the operation varied. In some cases gastro-jejunostomy was done, five out of nine cases so treated, dying. A good many cases had been treated by a modified form of Loretta's operation, and the mortality had not been great. Recently Mr. Dent had treated two cases of his (Dr. Cautley's) by pyloroplasty, an operation which was simple in the hands of a competent surgeon. It was done quickly, and the result was extremely good. In one case the child was fat and well, and had had no further symptoms. The other child died at the end of three or four months from the date of operating, from zymotic enteritis. The pyloroplasty had been quite successful, food passing readily through the pylorus.

Mr. H. J. Simson said he saw a case with typical symptoms of pyloric stricture, but the result of treatment was rather different. The child was first seen when five weeks old, and had been vomiting for a fortnight. The mother said the vomiting was as though the child was pumping the milk out, and everything it took was brought up soon afterwards. The child was constipated, and on tapping the abdomen peristalsis was seen passing from the left hypochondrium to the right, reaching down as far as the umbilicus. But, no thickening of the pylorus could be found, and it was recommended that the patient should be carefully dieted for a week, and if it did not get better it should be brought into hospital for operation. Small quantities of peptonised milk and water were given, and when seen a week later the vomiting had nearly stopped, but the peristaltic movement was quite visible on percussing the stomach. It was now being fed on ordinary milk and water. No peristaltic movement of the stomach could now be made out.

Dr. A. E. Sansom agreed with almost everything Dr. Cautley had said about the case. Something must be done, either in the way of better nutrition or of surgical interference. It seemed an excellent case for surgical interference.

Mr. A. H. Tubby said he would carefully avoid doing gastro-jejunostomy in the case. The difficulty, especially where the parts were very thin, was considerable, and the prospect held out by that operation was not good, so that it would introduce a new element of danger into the case. A great deal would depend on what was found when the abdomen was opened. He thought pyloroplasty would fully meet the case.

Mr. Donald J. Armour said if it was a case of congenital hypertrophic stenosis he did not see how anything short of operation could be of any benefit. He was present at the operation referred to by Dr. Cautley, who would agree that the stricture was very hard, and obviously nothing short of pyloroplasty could have reduced it.

Dr. George Carpenter said he could feel a definite thickening in the situation of the pylorus.

The Chairman (**Dr. G. A. Sutherland**) said all seemed to agree that dilatation of the stomach was present, but he thought there was possibly some obstruction lower down. He saw waves coming over the front of the abdominal wall, which appeared to be dependent on a contraction of the bowel beneath, but he did not think they pointed so entirely to obstruction of the pylorus. Evidently there was dilatation of the stomach, but he thought there was some obstruction lower down in addition.

Dr. Leonard Guthrie, in reply, said that when he first saw the case his impression was very strongly the same as Dr. Sutherland's, and that the obstruction lower down was in the nature of a partially constricting band. But, frequent subsequent examinations had convinced him that the peristalsis was of the stomach, not of the bowels. In all probability it was due to stenosis of the pylorus. Perhaps Dr. Cautley was correct in saying there was, in such cases, hyperplasia of the pylorus. With regard to treatment, Dr. Cautley had suggested that the child should be fed properly, but that had been carefully tried. The diet which suited the child best was one of whey and cream. If he were to send the child home it would certainly die, and it could not be kept in the hospital indefinitely, and therefore he was inclined to ask his surgical friends to assist him. The case was rather unusual, because the obstruction did not seem to be anything like so complete as it was in the case narrated by Dr. Cautley. He thanked Mr. Tubby for his observations; he proposed to leave to the surgeon who helped him in the case the decision as to procedure.

Postscript.—A few days subsequently to being shown at the Society's meeting, *pyloroplasty* was performed by Mr. John Murray. Considerable difficulty was experienced in feeding the infant for several weeks after the operation, as it seemed quite unable to digest milk in any form. Eventually, however, powers of digestion returned and the child made a complete recovery. In May, 1903, the mother wrote to say that the child was then in perfect health.

3. SPLENIC HYPERPLASIA: (1) VON JAKSCH'S DISEASE; (2) TUBERCULOUS.

By EDMUND CAUTLEY, M.D.

The patient illustrates that form of splenic enlargement which was described by von Jaksch in 1889 as a primary disease, under the name of anæmia infantum pseudo-leukæmica. By contrast with it, I shall detail the notes of another case in which the splenic hyperplasia was apparently due to a slow form of tuberculosis.

Harry J—, aged 13 months, was born at full time on September the 4th, 1901. He is the fourth child of a healthy mother, who had had one miscarriage between the second and third confinements. The eldest child is alive and well. Two are dead, one at three weeks of age and the other from measles. The patient has always lived in Battersea.

He was breast-fed for six months, given condensed milk for a month, and then had cow's milk and barley-water.

He came under my care at the age of 8 months, on May the 24th, 1902, with a history that he had been ailing for two months and had been weaned on this account. For one month a tumour in the splenic region had been noted and had increased steadily in size. He had been getting paler. The stools had been offensive, but there had been no diarrhœa or vomiting. On admission into the Belgrave Hospital for Children he weighed 13 lbs. 12 oz. The anæmia was marked but not severe. The skin had a somewhat yellowish tinge. Rachitic changes were present in the epiphyses and head, and there were no teeth. The liver was enlarged and firm, extending an inch and a half below the ribs. The spleen was relatively enormous, extending low down into the left iliac fossa and to the middle line, having a firm, well-defined, sharply rounded edge. The glands in the neck and groins were a little enlarged.

A blood-count on June the 4th showed a marked leucocytosis and some deficiency of erythrocytes, there being 89,000 white to 4,090,000 red cells, a proportion of 1 to 51.

A blood examination on June the 17th showed a great increase in the number of large lymphocytes. No myelocytes were noted, but the blood was not specially stained for this purpose, and possibly some of the large lymphocytes were really myelocytes.

On June the 25th the blood count was as follows :

Small lymphocytes .	50·1	per cent.
Large lymphocytes .	35·3	,,
Polynuclear cells .	9·8	,,
Eosinophiles .	4·7	,,

During the period he was in hospital he took food badly, was occasionally sick, passed offensive stools, had a little pulmonary catarrh, and slight epistaxis on two days. There was a variable, slight, irregular pyrexia, possibly due to the pulmonary catarrh or intestinal irritation. He was treated by dieting and by small doses of arsenic, and was discharged unimproved on July the 2nd, weighing an ounce less than on admission.

Three days later he was readmitted into the hospital, very much worse. He had lost a pound in weight, and had developed numerous petechial hæmorrhages over the limbs and back, and a few on the abdomen. The spleen was distinctly larger.

A blood examination on July the 14th showed a reduction in both white and red cells : white 40,000, red 3,260,000, a proportion of 1 to 81. Many myelocytes were present, also poikilocytes, megalocytes, and a few microcytes. Fresh petechial hæmorrhages developed for about two weeks, but in spite of this he slowly improved. He took and digested his food better, soon regained the weight he had lost, and slowly put on more weight. On August the 14th he was discharged much improved. The spleen was smaller, and he weighed 14 lbs. 3 oz.

From this date up to September the 27th he attended as

an out-patient and steadily improved. On September the 4th he was put on half-drachm doses of the syrup of the iodide of iron, three times a day. On September the 27th he weighed 15 lbs. 2 oz.

To-day it is noticeable that he is no longer anæmic. The evidence of rickets is still distinct. The liver remains the same size. The spleen reaches down to the iliac fossa, but it is much more freely movable, and is very much smaller in every dimension. The general health is good. The child is bright and cheerful, and takes his food well.

The age of the child, the clinical symptoms, and the course of the case are similar to what von Jaksch described. The only difference is in the blood examination, which never showed extensive oligocythæmia, and yielded a relatively high percentage of large lymphocytes. The blood count on July the 25th suggested a true lymphocytic leukæmia. The abnormally large number of myelocytes found during the second period of his stay in the hospital suggested that the case was one of myelogenous leukæmia, and the presence of numerous petechial hæmorrhages was also in favour of such a diagnosis. I am inclined to the view that the presence of the myelocytes was an indication of a sudden and profound alteration in the blood dependent upon the general health. Myelocytes are to be found in the cases described by von Jaksch, but not in such large numbers. The course of the case is opposed to a diagnosis of true leukæmia, and the variation in the blood type is, I believe, unknown in this disease.

A similar case was shown before the Society in the first year of its existence.*

Probably many varieties of splenic enlargement have been included under the name of anæmia infantum pseudoleukæmica. Some of them are cases of secondary anæmia with leucocytosis, of pernicious anæmia, of true leukæmia, of Hodgkin's disease or lymphadenoma, or of anæmia associated with rickets, congenital syphilis, or tuberculosis. In the present instance there is distinct evidence

* 'Reports,' vol. i, p. 154.

of rickets. The rickets is probably rather a concomitant than a cause. The frequent occurrence of even extreme rickets without marked enlargement of the spleen suggests that when such an enlargement does occur it is due to some other cause rather than the rickets. Again, congenital syphilis is common, but it is uncommon to find such large spleens in syphilitic infants, nor has post-mortem evidence been confirmatory of any such connection. It must not be forgotten that both rickets and inherited syphilis, and indeed any disease which interferes with nutrition cause changes in the blood, and tend to make it revert to the more infantile form in which there is a high proportion of lymphocytes and a low one of polynuclear cells. That great enlargement of the spleen may be dependent upon or associated with a tuberculous process is illustrated by the next case, which was under my care at the same time.

Willie K—, aged 14 months, was admitted into the Belgrave Hospital for Children on June the 9th, 1902, for left facial paralysis, otorrhœa, and enlarged glands in the neck; and he died on July the 17th. He had been an out-patient for diarrhœa and wasting during the previous June and July, and an in-patient for bronchitis during August. He had had no other illness, but was regarded as delicate. There was a history of phthisis on the mother's side. One brother had died from "consumptive bowels." Two other children were healthy. The glands on both sides of the neck were enlarged, movable, separate, hard, painless, and not adherent to the skin. Five teeth were cut. The fontanelle was small.

The percussion note was impaired over the right half of the manubrium sterni and in the first intercostal space for three fourths of an inch to the right of the sternum. The liver was much enlarged, reaching three inches below the ribs. The spleen extended down to the level of the crest of the ilium. No enlarged glands were felt in the axillæ or groins.

A blood examination on June the 9th showed moderate leucocytosis: white cells, 60,000; red cells, 7,470,000.

Small lymphocytes . .	14·4 per cent.
Large lymphocytes . .	31·5 ,,
Polynuclear cells . . .	53·9 ,,
Eosinophiles . . .	a few.

The child gradually got worse, developed rapidly progressive signs of consolidation in the right lung, and died of exhaustion. The course of the fever during the last three weeks was suggestive of tuberculosis, or septic infection.

Post-mortem examination.—The glands in the neck were beginning to break down, and contained some thin pus. A large mass of glands was found behind the manubrium sterni, and all round the roots of the lungs. Similar masses of glands were found in the mesentery. These glands were firm, white on section, and showed no sign of caseation. They resembled those seen in Hodgkin's disease. The right lung was slightly adherent. The upper lobe was solid, airless, mottled red and white in appearance. On squeezing, it ruptured in one place discharging thin pus, and a large cavity was found. The lower lobe was partly solidified. The left lung was unaffected. The liver was very large and pale. Sections showed extensive fatty degeneration, especially in the periphery of the lobules, the hepatic cells being converted into fat cells and there being comparatively little unaffected liver tissue left. The spleen was large, firm, and purplish in colour, and showed extensive fibrous hyperplasia. Neither in the liver, spleen, nor lungs was there any generalised or localised leucocytic infiltration, such as is seen in leukæmia and in Hodgkin's disease. On the other hand, in each of these organs there were found numerous large giant-cells, especially large in the spleen, and with a slight surrounding leucocytosis. No marked sign of caseous degeneration was found anywhere. Even in the spleen, where the process was most advanced, the tubercles seemed to be undergoing a fibrous change rather than a caseous one. The cavity in the lung, and

the breaking down of the glands in the neck seemed to be septic in character and not caseous.

The diagnosis in this case was extremely difficult. It might have been an instance of Hodgkin's disease, or of anæmia infantum pseudo-leukæmica, complicated by a secondary infection from otitis media. It is not uncommon for Hodgkin's disease to start as a secondary process, after a primary disease of the ear with glandular infection. Possibly it was a case of Hodgkin's disease, and the tuberculosis might be regarded as a tertiary complication. The post-mortem evidence negatives this diagnosis, but to the naked eye the appearance of the glands suggested lymphadenoma rather than tuberculosis. The microscopical examination showed that the case was one of chronic tuberculosis and secondary infection.

The blood examination showed a leucocytosis which was almost certainly inflammatory, modified by the age of the child in respect of the number of large lymphocytes. Compared with the first case, there was an actual increase in the number of red cells and not a diminution. Note also the difference between the relative size of the spleen and liver in the two cases.

It is not uncommon to find tuberculosis as a cause, or a complication of splenic hyperplasia, previously shown here (loc. cit.) In the discussion on a case, I referred to a case under my charge in which an exploratory operation had been done for enormous enlargement of the spleen with ascites. Extensive miliary tuberculosis of the peritoneum was present.

Many more observations are necessary to elucidate the different varieties of splenic enlargement in infants, and the characters of the blood associated with them. Care must be taken not to regard the reversion of the blood to the more infantile lymphocytic type as a sign of leukæmia. Care must also be taken not to ascribe to a primary disease conditions really due to a secondary infection. The field is a fertile one for the hæmatologist and pathologist. (*Exhibited October the 17th,* 1902).

Discussion.

Dr. Robert Hutchison said that as he had not had an opportunity of seeing the blood-films of the case, it was rather difficult to give an opinion. But, he thought it must be placed in that group of anæmias associated with splenic enlargement which was probably quite peculiar to children, and might be termed the splenic anæmias of infancy, which at one end of the series tailed off into mere slight splenic enlargement, and at the other developed into the extreme form of the disease to which von Jaksch gave the name anæmia pseudo-leukæmica infantum. Most people who gave attention to that matter thought it was not a morbid entity in itself, but was an extreme example of splenic enlargement associated with rickets and a certain amount of anæmia. The blood condition seemed to be one which was met with fairly often, a relative increase in the large mononuclear cells and a varying proportion of myelocytes. He had never seen more than 10 per cent. to 13 per cent. of the latter. Where there were a large number of myelocytes the cases did badly; but, Dr. Cautley's case seemed to be reaching a quiescent stage in which the blood condition was improved, and in which all that was left behind was a large spleen. Not long ago he had an example of that form of disease in which, although there was considerable rickets, the splenic enlargement was quite excessive, the spleen extending far below the umbilicus, but the blood changes were comparatively slight. Eventually the blood became normal, but the spleen and liver were enlarged, with a tendency to diminish. Microscopically, as far as he had seen, all that one could find was a general increase in the fibrous stroma, a condition which was common to many splenic conditions. There seemed to be no morbid histology characteristic of the disease.

Dr. A. E. Sansom said he was struck by the query with which Dr. Cautley ended his remarks as to the cause. The value of the Society was to bring together cases for which no interpretation seemed forthcoming. There must be a cause for the condition under consideration, but it was a very subtle one. It was all very well to put the case down as splenic anæmia, but there must be some cause for it. All one could say was that there was a perversion of blood-formation and of nutrition. The liver seemed to be getting normal, and it was the spleen which would cause anxiety in the future. Evidently the child had taken some poison into its system, which had produced what might almost be called a malarial intoxication. Probably there were many similarly difficult cases in the various hospitals, and it was necessary to go patiently to work.

Dr. C. W. Chapman asked whether such cases were commoner among poor people than among the more fortunate class.

The Chairman (**Dr. G. A. Sutherland**) asked Dr. Cautley's opinion as to the prognosis in such cases. He believed it was thought to be fairly good, if the child did not succumb to some intercurrent disease to which he was very liable in consequence of his weakness. In the present week he had seen a seven-year-old child who suffered from typical splenic anæmia when it was twelve months old. He remembered treating that child very energetically, but apparently without any benefit. From the date of the cessation of treatment, however, it steadily improved, and had done extremely well. At the present time its spleen was not in any way enlarged. He had had a like experience in other cases. Of course one treated the case constitutionally and regulated the diet. He agreed with Dr. Sansom that the most likely ætiology was that some poison was absorbed into the system which produced effects there, those effects lasting for a certain time and then passing off entirely.

Dr. Edmund Cautley, in reply, said he had seen no cases among the better classes, but one saw much more of the poorer classes, particularly in hospital. Apart from that, he would not expect to find the disease as prevalent among richer people, because the children would be brought for treatment earlier. In his experience, the prognosis was fairly good. The usual history was that the child went out of hospital after a certain period and then disappeared, no more being heard of it. The cases which had died in hospital nearly always had some other condition associated with them. In one case, where the spleen was as large as in the present patient, he had the abdomen opened to see if it was possible to take the spleen away, particularly as there was fluid and the child was very ill. As soon as the abdomen was opened one found, in addition to the enormous spleen, general tuberculous peritonitis. That child died immediately the spleen was handled. Another case which apparently was identical with the present, except with regard to the blood, was under his care, and that child died. On examining the spleen there was distinct evidence of tubercle. There was generally some complication to account for death in such cases. He agreed that general treatment was best. The present child had improved most under the syrup of iodide of iron. He was interested in Dr. Hutchison's remarks about myelocytes. When the blood was first examined very few were found, but the second time the child was admitted a different stain was used at the examination, and a great number of myelocytes were detected. At that time the child was very ill indeed; it had numerous petechial hæmorrhages, and had gradually lost weight. But still the child had recovered, which seemed to show that many myelocytes did not necessarily indicate a fatal prognosis.

4. HYDROCEPHALUS ARRESTED BY REPEATED LUMBAR PUNCTURE.

By EDMUND CAUTLEY, M.D.

Lily O— was born at full-time in July, 1901, and came under my care in March, 1902, at the Metropolitan Hospital.

She was the first child by the second husband. The mother had had six children and six miscarriages by a previous husband. She had been partially breast-fed, and began to waste at six weeks of age. At five months old the head became retracted, and the child was irritable. She remained in much the same condition up to the date of admission into the hospital on March the 13th, 1902. She was then eight months old, and only weighed 8 lbs. 1 oz., being much wasted, drowsy, and having a retracted head. The head was 17 inches in circumference, the anterior fontanelle was large and tense, the sutures were open, and there was no craniotabes. The eyes were normal, the teeth were absent, the palate was abnormally high, and there was moderate nasal catarrh. The ribs were beaded, the arms were rather rigid, and the legs were extended and rigid, the knee-jerks being exaggerated.

A diagnosis of hydrocephalus, secondary to a basal meningitis, was made, and on the assumption that the latter might have been syphilitic, mercury and iodide of potassium were prescribed. The diet consisted of peptonised milk.

On March the 21st half an ounce of cerebro-spinal fluid was drawn off by lumbar puncture. The fluid was clear and sterile.

During April the spinal canal was tapped in the lumbar region four times, from 1½ to 3 ounces being drawn off at a time, a total amount of 9½ ounces. The child gained 16 ounces in weight, took food better, and was more comfortable. Both patellar and plantar reflexes were exaggerated.

During May she was tapped four times, from $1\frac{1}{2}$ to $2\frac{1}{2}$ ounces of fluid being drawn off at a time, $7\frac{3}{4}$ths ounces in all. She gained 20 ounces in weight, in spite of an attack of measles, the rash of which came out on May 18th, and which was followed by otorrhœa a week later. The diet was increased by the addition of Benger's food.

During June she was tapped four times, from $2\frac{1}{8}$th to $3\frac{1}{2}$ ounces being drawn off at a time, in all $10\frac{3}{8}$ths ounces. She gained 40 ounces in weight, seemed better in every respect, and the retraction of the head and rigidity of the limbs were almost absent.

During July she was tapped three times, and 4 ounces of fluid in all were drawn off. She gained 35 ounces in weight.

On August the 6th she was tapped for the last time, and 2 ounces were drawn off.

To sum up :—She was tapped on seventeen occasions, from March the 21st to August the 6th, and cerebro-spinal fluid to the amount of $34\frac{3}{8}$ths ounces was drawn off. During this period the child gained 124 ounces, in fact almost doubled her weight, in spite of an attack of measles. The average rate works out at 6 ounces a week. It was very noticeable that after nearly every tapping the child rapidly put on weight, in spite of the withdrawal of fluid, and seemed better in every respect. The most marked gain was one of 16 ounces from June the 6th to June the 13th, during which week she was tapped twice and 6 ounces of fluid were drawn off. During the next week she was tapped once and gained 18 ounces.

Very great credit is due to my house-physician, Dr. F. Norman White, who carried out the treatment with the greatest of care and skill, and without causing a single bad symptom. In no case was the fluid withdrawn by aspiration, it was allowed to flow out of its own accord. Sometimes it would go on trickling for as long as half an hour.

The tapping was stopped for two reasons. In the first place, there ceased to be evidence of increased intra-cranial pressure, as indicated by a tense fontanelle. The

fontanelle remained soft and the sutures had all closed up. Secondly, it was thought that no benefit would result from further tapping, in view of the general condition of the child, and the fact that the fluid during the last few tappings had not flowed as readily.

Since the tappings were omitted the child had gained weight, but at a less rapid rate, namely, a little over 3 ounces a week. She had cut six teeth during this time without any trouble.

In spite of the withdrawal of fluid, and the absence of tenseness of the fontanelle during the last two months, the head has slowly increased in size. The increase is greater than can be ascribed to the growth of the child, who is now fifteen months old, weighs 17 lbs. 13 ounces, and has a head measuring 21 inches in its greatest circumference. The head is clearly hydrocephalic, although the fontanelle is small and soft.

The cerebro-spinal fluid was always clear, sterile when it was examined bacteriologically, and devoid of morphological elements. For the latter, the sediment was examined microscopically after the fluid had stood for twenty-four hours in a conical glass. It was not centrifugalised. At times it contained a very faint trace of albumen, and a slight but variable amount of a substance which reduced copper salts.

The present state of the child is not satisfactory. The head is large, heavy, cannot be supported by the child, and is somewhat retracted. Apparently she is blind, or has merely a slight perception of light, although the optic discs show no change. She is not deaf and to a certain extent recognises her nurses by the voice. She eats and sleeps well, but her mental condition shows no improvement and has, I think, somewhat deteriorated during the last month.

When first admitted into the hospital she was in a most precarious state, marasmic, taking food badly, and at times vomiting. There is no doubt that the frequent tapping delayed the progress of the effusion of fluid, kept the child

alive, and allowed her to grow and gain weight. At present a state of equilibrium seems to have been established. Unfortunately, the treatment has not been sufficient to effect a cure, and the result is by no means a good one. Possibly, if more fluid had been drawn off, and the treatment had been continued for a longer period a better result might have been attained.

The theory on which I adopted this line of treatment was based on the assumption that the hydrocephalus was developing slowly, as a result of basal meningitis. At first there was little evidence of excess of fluid in the ventricles. I thought it possible that the frequent removal of small quantities of fluid would prevent the progress of the hydrocephalus, would allow the cerebral conditions to accommodate themselves, would keep the child alive, would permit it to gain flesh and strength and a better state of nutrition generally, and thus assist in a more healthy state of nutrition of the cerebral tissues. Certainly the child has thrived, remarkably so, but no permanent benefit has resulted from the treatment.

It must not be forgotten that secondary hydrocephalus may cease to increase of its own accord, when it has reached a certain stage. We must not, therefore, lay too much stress on the benefits to be derived from lumbar puncture, and ascribe arrest of effusion to an occasional tapping. The present case shows that the treatment can be carried out successfully for some months without producing bad symptoms, and that it did actually keep a child alive who otherwise did not seem likely to live more than a few weeks.

(*Exhibited October the* 17*th*, 1902).

Discussion.

Dr. Robert Hutchison said lumbar puncture was a method of treatment which had been suggested in America, where it was recommended not merely as a diagnostic, but also as a therapeutic measure. He had tried it on his own cases, but his results had been much more disappointing. In two of the children, after the fourth tapping a sudden and fatal collapse came

on, and he could not help thinking there was a causal relationship between the tapping and the collapse. He had seen cases in which permanent drainage had been established in the lumbar region, and in those cases there seemed to be a sort of tumbling-in of the brain when the fluid was withdrawn. That was a danger which should be borne in mind. It was an interesting fact that such cases could be tapped from the lumbar region, because that at once disposed of the popular idea that in such a condition there was a blocking at the base of the brain. Even when such success as Dr. Cautley had was attained, it was unfortunate that the ultimate result was so bad: the children were nearly always blind and imbecile. If tapping could be commenced earlier possibly the blindness could be prevented. In spite of his bad results, he would treat his cases by tapping early and tapping often.

Mr. F. N. White (introduced) said he was Dr. Cautley's house-physician and drew off the fluid. He used an ordinary exploring needle, the fluid came away very slowly, an ounce of fluid requiring about half an hour. There was never any complication of any sort.

The Chairman (**Dr. G. A. Sutherland**) said he had seen the accounts of the German and American cases which had been treated by puncture, and he understood that that treatment had been given up in America and Germany as futile. The experience he had had in hydrocephalus was that usually a closure existed about the base of the brain, so that there was no possibility of communication down the spinal meninges. Therefore, he had come to regard that as the pathology of cases of hydrocephalus following cerebro-spinal meningitis in infancy, what was described as posterior basal meningitis. He did not intend to try repeated puncture, as Dr. Cautley had done in his case. Was it possible that a similar amount of fluid could be drawn from a normal spinal canal? He (Dr. Sutherland) had had a similar experience to Dr. Hutchison, sudden death occurring almost immediately after withdrawing the fluid. Had Dr. Cautley any evidence of an accumulation of fluid in the spinal canal?

Dr. Edmund Cautley, in reply, said he had one case of hydrocephalus in which the spinal canal was tapped and ½ an ounce to 1 ounce of fluid was withdrawn. On a second occasion it was punctured and no fluid was obtained. The child died, and no excess of fluid was found in the spinal canal, and no communication with the cerebral ventricles. He thought it very unlikely that 3½ ounces of fluid would be found in the normal spinal canal of a child ten months old. He thought the treatment carried out was likely to be successful in only a few cases. The child came under treatment three months after the symptoms of basal meningitis occurred, and the head was not enlarged.

5. MICROCEPHALUS WITH UNUSUAL SYMPTOMS.

By EDMUND CAUTLEY, M.D.

Fred B—, aged 4 years, was admitted into the Belgrave Hospital for Children, under my care, on August the 21st, 1902.

He was born in October, 1898, and up to the age of twenty-eight months was well and strong; he could walk and run, but was unable to talk.

He was one of seven children, of whom two are dead. There was nothing in the family history indicative of inherited syphilis, or of nervous affections. He was a full-time child, and breast-fed for eight months. At six months he had one "fit," but has had no more until recently. He had varicella two years ago and measles last summer.

Weakness of the legs was first noticed twenty months ago; he "used to fall about." Gradually this weakness increased, and for eight months he has been unable to walk. At the beginning of August he got much worse. The weakness increased, he could not even sit up, and he began to have fits. A week later he had difficulty in swallowing and could not take solid food. The fits were then frequent and lasted for about half a minute.

On admission it was evident that his intellect was impaired. He did not talk, cry, or laugh. He was unable to swallow, and had taken no solid food for two weeks, and he had to be fed by a nasal tube. He passed everything under him. He could not sit up in bed, or hold up his head. He could not move his legs, and only to a slight extent used his arms and hands in an incoordinate manner, the left more than the right. The head measured 18 inches in circumference. There was no squint, the pupils were normal, and there were no changes in the fundus oculi.

Examination of the muscular system showed complete loss of power in the lower limbs, back, and neck, and an almost complete loss in the arms. The muscles were flabby, and there seemed to be a certain amount of general wasting, which was probably due to disuse and insufficient food. The electrical reactions were unaltered. Sensation seemed a little blunted, on application of a fairly strong faradic current. The knee-jerks were deficient, probably on account of the bromide which he had been taking for the fits. An extensor plantar reflex was obtained, and there was no ankle-clonus.

The fits were numerous, ten to twelve in the twenty-four hours, general and epileptic in character. During the fits the face became congested, the arms and legs rigid, the body bent towards the right side, and the eyes turned upwards to the right. They were followed by relaxation, pallor, and unconsciousness for about half a minute, and once for two minutes.

On August the 29th he was much brighter, did not require nasal feeding, and could pull himself up in his cot. The fits were fewer in number and milder in character. The knee-jerks were active.

On September the 28th it was noted that his condition had remained about the same, though it had varied a little from time to time. Twice he had been definitely worse, having more fits, and requiring nasal feeding. Now, he had improved, and had only two or three fits in the twenty-four hours. The knee-jerks were active, and there was no extensor plantar reflex. At present he shows definite signs of improvement. He can sit up a little in bed, seems more intelligent, and expresses emotion by trying to cry. He has only one fit daily, which usually begins with a loud harsh cry. No nasal feeding has been required for two weeks. He still passes everything under him.

The case is somewhat mysterious in its characteristics and course. As a basis for diagnosis we have the small head, the fits, the impaired intellect, the marked loss of

muscular power, the absence of muscular degeneration, and the presence of active knee-jerks.

The course is peculiar in two respects. First, there is the gradual loss of muscular power, with the later development of fits and imbecility, in a child who was an apparently normal child, except for backwardness in learning to talk. Secondly, we must note the definite improvement under treatment.

The loss of power is apparently dependent upon the central nervous system and is cerebral in origin, for there is no reaction of degeneration and no spasticity.

A slow-growing intra-cranial tumour can be excluded by the fact that the child is improving, and by the absence of the usual symptoms, such as headache, vomiting, and optic neuritis, and by the absence of any localising symptoms.

A possible diagnosis is primary degeneration of the pyramidal cells of the cortex of the brain, due to some inherent defect, or the action of some toxin. This seems a reasonable view, in spite of the absence of a history of any exciting cause. The improvement under treatment may be due to improved nutrition from better feeding.

That the child may be called a microcephalic idiot at the present time is perfectly true, and perhaps this explains the whole condition. In my experience, however, the sequence of symptoms is one I had never met with in microcephalus. I have not come across a microcephalic idiot who has been able to walk, and has then gradually lost the power. It seems to me necessary to conclude that there is some additional factor, and the sequence of symptoms suggests that the case is possibly one of cerebral degeneration occurring in a microcephalic child. No matter what the actual diagnosis may be, the prognosis is undoubtedly a bad one. We can hardly hope that the boy will completely regain normal intelligence.

(*Exhibited October the* 17*th,* 1902.)

6. A CASE OF DEFORMITIES.

By FRANK DEAS.

The patient shown was a male child, aged 1 year and 7 months; it had the following deformities.

Right hand.—The thumb was normal. The index and middle fingers were united by thick fleshy union as far as first interphalangeal joint. The middle and ring fingers were united throughout their whole extent at birth by a

Fig. 1.

thin skin web; this had been cured. The little finger was absent.

An X-ray photograph showed only three metacarpal bones—one for the thumb, one for the ring finger, and a middle one for the fused index and middle fingers.

The left femur was one and three quarter inches shorter than the right. An X-ray photograph showed the bone to be shorter and smaller than that of the right.

(*Exhibited November the 21st*, 1902.)

Discussion.

Mr. A. H. Tubby said the Society was indebted to Mr. Deas for bringing the case forward. If Mr. Deas was present at the birth, he (Mr. Tubby) would like to hear whether there was a large amount of liquor amnii present, and therefore whether there was much packing *in utero*. Also, what was the length of the umbilical cord? The books also described in such cases an amniotic band, but he had not sufficient obstetric experience to know whether such existed, though he had doubts on the matter. With regard to operating on such a hand, there were three metacarpal bones and four fingers, and he thought the best result would be obtained not by attempting to separate what were presumably the first and second fingers, but by amputating half of one and using the skin to recover the remaining half. His own experience in such cases was getting somewhat large, and he found that when mesoblastic tissues (muscle or tendon) united two fused fingers the results were not good, whether Thiersch grafts, or other classical methods were used.

Mr. Deas (Merton) thanked Mr. Tubby for his advice and, in reply to that gentleman's questions, said he was not present at the birth, which had taken place forty-five minutes before he arrived.

7. A SYPHILITIC LIVER (GUMMATOUS) FROM A GIRL OF TEN YEARS.

By HENRY ASHBY, M.D.

The specimen shown was a lardaceous liver containing gummata, from a girl aged 10 years. The patient had been under observation for four or five years with a hypertrophic condition of the bones of both forearms, and also the tibiæ.* At the post-mortem examination the kidneys were "large white" and lardaceous, and the liver and spleen were also lardaceous; the liver was in an extreme lardaceous condition. There were fifteen or sixteen gum-

* One of the cases shown at the Manchester Provincial Meeting (*vide* 'Reports,' vol. ii, p. 247, Case No. 8).

mata the size of small nuts visible on the surface, and also seen on section. There were several scars on the surface.

Fig. 2.—Liver showing gummata and localised perihepatitic scars.

Professor S. Delépine was good enough to confirm by microscopical examination the gummatous nature of the lesions.

(*Exhibited November the* 21*st*, 1902.)

Discussion.

The Chairman (**Dr. A. E. Sansom**), in thanking Dr. Ashby, said he could not clearly recall the child at the Pendlebury Hospital, Manchester, when the Society visited that city, because the wealth of material provided was so great; but the case was one of extreme scientific value.

Mr. D'Arcy Power asked whether the joints were examined, and whether any joint trouble was complained of by the patient. In one or two syphilitic cases he had seen, the joints were considerably affected, though during life there was not much evidence of it. It would be interesting to hear whether the thickening of the bones was due to periostitis.

Mr. A. H. Tubby asked whether the curious condition

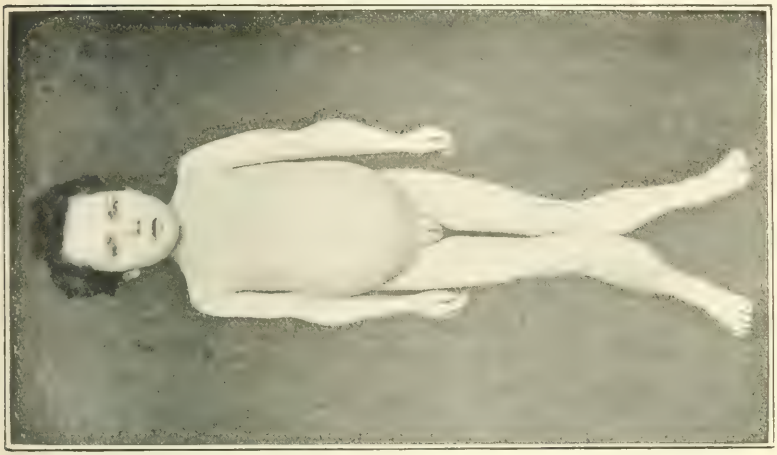


FIG. 4.

FIG. 4. - Four sections of the liver. The first two are sections of the right lobe, the last two are sections of the left lobe. One-half of the third section and the whole of the fourth section were stained with a solution of iodine to show the extent of the lardaceous infiltration.

known as pneumonia alba—syphilitic infiltration of the lungs—was present in the case shown by Dr. Ashby.

Dr. Theodore Fisher (Bristol) showed a sketch of a case under his care, which clinically somewhat resembled Dr. Ashby's. The child was first seen at the age of six years, and its liver was felt to be enlarged and deeply scarred, like the syphilitic liver of the adult. The first child in the family was born alive; then there were three miscarriages; next the boy he was referring to was born, and following him were twins who lived a month; then there was a miscarriage, and finally a boy who had lived fifteen months. The boy to whom he first alluded died three years later, and the liver was found, post mortem, to be deeply scarred in the situations of former gummata.

Mr. Clement Lucas said it would be interesting to hear what was the experience of the members upon the treatment of the bone affections which were so prominent in Dr. Ashby's case. His own experience of chronic ostitis due to hereditary syphilis was, that it was an incurable affection. He had watched a case which had chronic ostitis of the tibia, and another with chronic ostitis of the femur, for as long as ten or fifteen years. He could not say that his treatment by the local application of mercury, and the administration of both mercury and iodide of potassium internally had any obvious effect in reducing the chronic ostitis, when it had once decidedly developed. Many years ago, as a junior assistant-surgeon, he amputated one such case, because it was thought by his senior colleagues that the case was one of tumour of the ulna. There was an enormous thickening and, even when cut down upon, it was thought to be a tumour; and he was particularly impressed by his colleagues on the necessity of removing the enlarged glands which were above the part. It was really a case of syphilitic ostitis, which had previously been under treatment for a long time with mercurial inunction, and mercury and iodide of potassium internally. His further experience of such cases confirmed his belief that they were the most chronic forms of ostitis which could be met with. He had incised bones, done linear osteotomies, and chiselled out portions of bone, but no treatment seemed to be satisfactory. He would be interested to hear whether Dr. Ashby's surgical colleagues had been consulted about the case, and whether they could give any hope in cases where there was very great enlargement of bone.

The Chairman (**Dr. A. E. Sansom**) thought the case narrated and illustrated by Dr. Theodore Fisher was scarcely of the same nature as Dr. Ashby's, which was demonstrably gummatous.

Dr. Henry Ashby (Manchester), in reply, said that although the girl was under treatment four or five years, neither the gummata nor the bone lesions were cured. He thought all would

agree with Mr. Lucas that the bones in such cases were very little influenced by either iodide of potassium or mercury. The girl was taken into the hospital from time to time, when there were tender nodes on the shins, and then, by giving rest and medicines, they were brought down to a chronic state. With regard to the state of the bones, permission could not be had for obtaining them. In the lungs there was only hypostatic trouble.

8. CICATRICIAL CONSTRICTION OF THE ABDOMINAL WALL AND OF THE LEFT THIGH, ATTRIBUTED TO COMPRESSION BY THE UMBILICAL CORD.

By C. O. HAWTHORNE, M.D.

The unusual conditions existing in this case are clearly displayed in the accompanying illustrations. As is shown in the figures, there is a narrow cicatricial band forming a complete girdle of constriction in the abdominal wall. Behind, it is placed at the level of the third lumbar spine; on each side, it passes forwards and slightly downwards just above the level of the iliac crest; and the circle is concluded in front at a point 1¼th of an inch below the umbilicus. The cicatrix appears to involve only the superficial part of the skin, but it grips the abdominal wall to such an extent that it produces a groove, which in the anterior part is depressed from ¼th to ½ an inch below the surface. Posteriorly, there is some appearance of a double groove, but this cannot be traced in the lateral and anterior parts of the circle.

The lower part of the left thigh also shows a depression or furrow placed obliquely immediately above the knee. The skin here shows no distinct cicatricial change, and the constriction is much less acute than that in the abdominal wall.

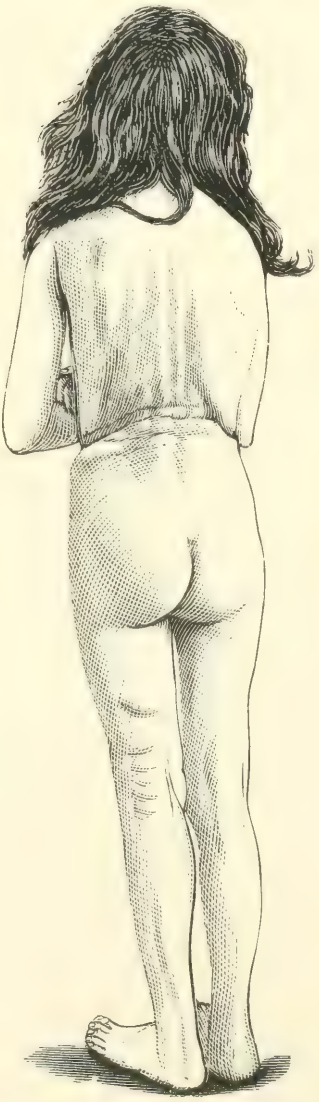
These conditions have, without doubt, been present from birth. Unfortunately, it has not been possible to trace the practitioner who was in attendance when the child was born—she is now ten years of age—and therefore expert

testimony in reference to the then existing facts is not available. But, according to the mother's statement, delivery took place at the seventh month, and the grooves on the abdominal wall and thigh were occupied by the umbilical cord. The same witness credits the doctor with the remark that "had the pregnancy gone to full time the child would have been cut into three parts."

The chief feature of interest in the case is the question of the agency by which the grooves on the abdominal wall and thigh were produced. It may be assumed that at the time of birth these depressions were certainly occupied by the umbilical cord, because, apart from the mother's statement as quoted above, other cases have been recorded in which a loop of the cord has been found in somewhat similar constrictions, at least in the case of the limbs. Thus, it may be fairly argued that there is a *primâ facie* case in favour of the umbilical cord as the constricting agent. But, the evidence is not quite conclusive. For it must be allowed, that given during intra-uterine life the formation of grooves upon the surface from any cause whatsoever, the mechanical conditions are such as would lead more or less readily to the reception and retention of the umbilical cord within such depressions. It is therefore possible that occupancy by the cord may be not the cause but the consequence of these constrictions. Hence, it must be considered whether there is any other feasible constricting agent. First of all in this respect comes the possibility of bands of tissue taking origin in connection with the formation of the amnion. These are usually regarded as agents which may lead to intra-uterine amputation of the digits or limbs, and some instances have been recorded in which they have been found constricting these parts. All that can be said in the present case is that there is no evidence of the existence or action of such bands. In the second place, there is the suggestion that constrictions and amputations of the limbs occurring during intra-uterine life are due not to some external agent, but to a trophic or sclerodermic change, such change not only involving the skin, but cutting

FIG. 5.

FIG. 6.



its way, as it were, more or less deeply into the tissues and, in the most extreme instances, causing strangulation, or even separation of the distal part. Dr. J. W. Ballantyne, who discusses this question in his latest work on 'Antenatal Pathology,' considers that no "satisfactory explanation of the production of the so-called spontaneous amputations has yet been advanced." In view of so authoritative a statement, some reserve in the expression of an opinion is necessary, but it may be permitted to suggest that in the present instance there are facts which support the view that here, at all events, the umbilical cord is responsible for the existing conditions. These are found more particularly in connection with the groove on the left thigh. This, it will be observed, has not a circular, but a spiral direction and form. Now, whilst it may be conceivable that a trophic, or sclerodermic lesion may produce a circular constriction of a digit or limb, it is less easy to imagine an oblique or spiral groove, such as is here represented, to be the result of such a lesion. Further, the skin forming the floor of this groove is apparently normal in every respect; the appearances are just what might be expected from prolonged compression by a broad, smooth ligature. Add to these facts the further fact (as it may be assumed to be) that the cord was found in the groove at the time of the child's birth, and the evidence supporting the theory that the cord is responsible for the existence of the groove is, it must be admitted, very strong, if not actually conclusive. But, if this view holds good in reference to the thigh it must apply with almost equal force to the constriction of the abdominal wall. It is only necessary to suppose a greater degree of tension to explain both the more marked depression, and the definite cicatricial change. This hypothesis also accommodates itself to the appearance of a double groove on the posterior wall of the abdomen with the existence of a single furrow in the rest of the circle. For, in the former position the firm structure of the wall opposes a deep depression, and thus a limited play of the cord from one level to another becomes possible;

whereas the yielding nature of the lateral and anterior abdominal walls favours the ready formation of a deep and narrow furrow, along the line of which the compressing action of a tense cord would be continuous. Such a conclusion bears upon the question of the causation of intra-uterine amputations. That the agency producing these is identical with that producing constrictions of the limbs is fairly certain. For, in addition to the common-sense presumption that amputation is merely constriction carried to an extreme degree, there is the observation that both conditions may be present in one and the same child. An example of this association was recently shown at the Polyclinic, the case being that of an infant born *minus* several fingers, and with a deep constricting groove around one leg. But, if intra-uterine amputations are, or may be, due to the same cause as that which produces constrictions of the limbs; and if, as is rendered in the highest degree probable from the present case, such constrictions may be caused by the umbilical cord acting as a constricting agent, it follows that intra-uterine amputations may also be accomplished by the cord, or other band external to the fœtus. Hence, the present case may be advanced as a contribution to the discussion regarding the mechanism of intra-uterine amputation. And its influence goes to show that such amputations are caused by an external band constricting in an extreme fashion the affected part. The argument, in brief, is that intra-uterine amputations and constrictions of the limbs are produced by one and the same agency; that, as is here shown, such constrictions may be caused by an external constricting band, in this instance the umbilical cord; and that, consequently, such bands must be held responsible for the occurrence of intra-uterine amputations.

(*Exhibited November the 21st*, 1902.)

Discussion.

The Chairman (**Dr. A. E. Sansom**) said if the ligature theory advanced was well-founded it was fortunate that the birth took place at the seventh instead of at the ninth month. The explanation given seemed a reasonable one.

9. BILATERAL COLOBOMA OF THE IRIS, CHOROID, AND OPTIC DISC.

By C. O. HAWTHORNE, M.D.

This is an example of a congenital abnormality, namely, imperfect closure of the fœtal ocular cleft. The defect is not, as in the more frequent cases, limited to the iris. It involves also the choroid and the optic disc. A further measure of interest is provided by the fact that the defect exists, and to an equal extent, in the two eyes. It is the extreme development and symmetry of the condition which are the special features of the case. The lens of the right eye has recently become opaque, but when first seen, some twelve months ago, the right fundus showed a condition exactly parallel to the facts present in the left eye.

As points of detail, it may be noted that the coloboma in each iris is not a gap gradually narrowing from the pupillary margin to the circumference of the iris; it has, on the contrary, the appearance of a rectangle cut out of the iris substance, so that the coloboma and pupil together have the shape of a keyhole. Again, across the base of the coloboma in the left iris is a strand of iris tissue separating it from the opening of the pupil, and justifying the term "bridge-coloboma." A third fact is the existence of a notch in the lower margin of the left lens—the so-called coloboma of the lens. In all respects other than the above the eyes seem to be of normal development.

The patient, a girl of thirteen years, is, with the exception of her eyes, well-developed and free from bodily deformity; she is one of a family of seven, and neither in the present, nor in the earlier generations is there any history of peculiarity in the appearance of the eyes, or of defective sight.

(*Exhibited November the 21st*, 1902.)

10. SUBCUTANEOUS TRAUMATIC RUPTURE OF A SPINA BIFIDA.

By R. C. DUN, C.M.

This case of spina bifida is one in which there was, apparently, subcutaneous rupture of the sac, the result of an injury.

The patient is a girl of six years old. I first saw her in the out-patient department of the Children's Infirmary, Liverpool. The mother, a careful and intelligent woman, gave me the following history. The child was born with a swelling low down on the back, covered with thin red skin, which afterwards became white. The swelling kept pace with her growth. There was never any complaint of pain. She always made water more frequently than the other children, and habitually wetted the bed at night.

Six weeks before the girl was brought to me she was run over by a light milk-cart. The driver of the cart took her to one of the hospitals, where she was examined, and the man afterwards drove her home. He distinctly told the mother that one wheel had passed over the child.

On undressing her the mother noticed that the swelling on the back had entirely disappeared. The clothes were not wet.

She was kept in bed for a week; from the time of the accident she always had pain when any pressure was made over the lower part of the spine, and she could not lie on her back.

About a fortnight after the accident a swelling was noticed on the buttock to the left side; this gradually increased in size, and four or five days later the old swelling in the middle line gradually began to reappear. Since then both swellings have grown larger.

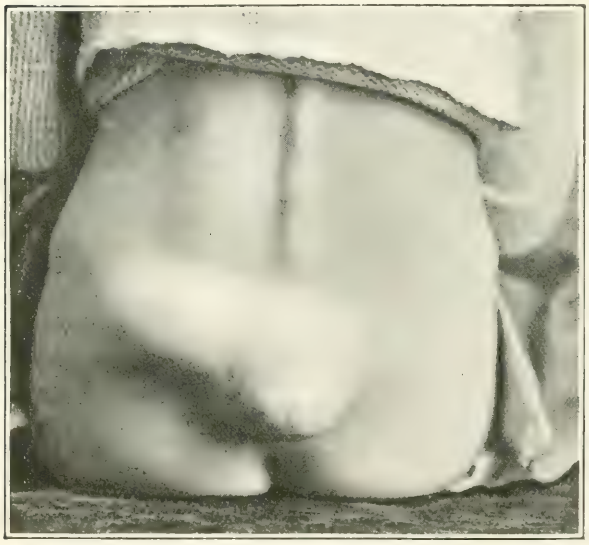
For a few days after the accident there was complete incontinence of urine and fæces; this loss of power was recovered from gradually.

The child was brought to the hospital on account of the pain which was present on pressure over the swellings on the back, and their progressive increase in size.

On June the 21st, 1902 (eight weeks after the accident) the child was admitted into the Children's Infirmary, and the following notes were taken.

Both swellings on the back have considerably increased

Fig. 7.



in size since she was seen in the out-patient department fourteen days ago.

In the middle line of the back, over the last lumbar vertebra and upper three pieces of the sacrum, there is a swelling bluntly conical in shape, rounded at its base, and covered with normal skin, except towards its apex where there is white cicatricial tissue. From the normal skin over the tumour there is a plentiful growth of fine short hair. Projecting outwards and to the left from the side of this first swelling there is a second swelling, flatter and

longer and ending in a rounded extremity. Its lies transversely over the left dorsum ilii. At its right end it merges with the left side of the larger conical swelling in the middle line. There is a slight sulcus where the two tumours meet; hairs are present in this groove, but do not extend over the swelling to the left. On palpation, both tumours fluctuate distinctly, the fluid wave passing freely from one to the other. The edge of both is ill-defined. No bony deficiency can be felt. There is considerable pain on palpation, causing the child to cry out. On firm pressure with the flat of the hand on the swelling to the left, it can be made to disappear almost entirely, the large right tumour becoming increased in size and tension. On pressing thus, the left posterior superior spine can be distinctly felt.

The child is unable to lie on her back, owing to the pain which she suffers in this position.

She micturates about every two hours during the day, and has dribbling of urine at night. The bladder is not overdistended, the urine is normal. She has complete control of the bowels. There are no deformities. There are no paralyses, anæsthetic areas, or trophic lesions of the lower extremities.

On June the 30th I operated. I made two vertical incisions, slightly curved to surround the cicatricial area on the median swelling, and passing well above and below it. Cutting through a small amount of areolar tissue I got down upon the left side of the main sac, which was thick and strong, and proceeded to peel off the superficial structures from it, which was easily accomplished. On reaching the sulcus between the two tumours I came upon a very thin translucent sac passing out from the side of the thicker one. I exposed this for about an inch from the central swelling; it was firmly adherent to the overlying subcutaneous tissue, and was defined with difficulty. I then made a half-inch vertical incision into it. Clear fluid escaped. On enlarging the opening, and raising the edges of the thin sac with forceps, I saw a round

opening rather larger than a threepenny piece, which led into the thicker central sac. On inserting the finger, the edges of this opening felt firm and resistent, and perfectly smooth. The finger could be passed outwards in the thinner sac for 2½ inches; the whole wall was smooth. I next opened the central sac by a laterally placed vertical incision on the left side, and found it to be about four times the thickness of the lateral one previously opened. Numerous strands of nerve tissue could then be distinctly seen passing from the deficiency in the spinal canal on to the sac. The skin was next thrown off the right side of the tumour, the sac opened on that side, and the nerves dissected away from the sac. In this I was only partially successful, for two strands of nerve tissue were so firmly adherent that I was forced to desist in my efforts of separation, and to return them to the spinal canal with thin strips of sac attached. The remainder of the thick sac was then cut away, and the edges inverted and united with a continuous silk suture. Two lateral musculo-fascial flaps were cut, and also sutured with inverted edges over the bony gap.

I now attempted to dissect out the thin laterally placed sac. This I found was a matter of great difficulty, as the wall was very thin and firmly adherent to overlying tissue. It tore readily on picking it up with forceps. After getting away small portions, I thought that the time spent was unnecessarily prolonging the operation, and serving no useful purpose; so I left the remainder, and closed the superficial wound.

The after progress as regards her general condition was uneventful.

For three days there was complete incontinence of urine and fæces. On the fourth day the control of the bowel began to return, and was complete on the seventh day.

The urinary condition was slower in improvement, but in three weeks it had returned to the state which existed before the operation, viz. micturition every two to three hours during the day, with dribbling at night.

There was no pain after the operation, and the child can now lie comfortably on her back, and experiences no discomfort on pressure on the cicatrix.

I had hoped that my operation might improve the partial bladder paralysis, but my inability to separate all the adherent nerves from the sac probably accounts for my failure in this respect.

The clear history of the sudden disappearance of the old spina bifida immediately following an injury, without any external escape of fluid, the subsequent appearance of a fluctuating swelling in close relation to the position of the original one, and a later distension of the old sac, all point to subcutaneous rupture of the spina bifida which had existed since birth.

I take it, that on rupture of the sac the cerebro-spinal fluid was forced into the areolar tissue over the left gluteal muscles, that gradually it became encapsuled, and further leakage from the spinal canal caused the appearance of the laterally placed swelling. When the full capacity of this false sac was reached distension of the old one followed, with reappearance of the original spina bifida.

I think that the difference in thickness of the two sacs, the close adherence of the areolar tissue to the laterally placed one, and the small sharp cut opening between the two, all bear out this view. (*Read November the 21st,* 1902.)

11. ECTOPIA OF THE BLADDER WITHOUT DIVISION OF ITS WALLS; PARTIAL EPISPADIAS; INCOMPLETE DESCENT OF THE TESTICLES.

By R. C. DUN, C.M.

The patient is 6 months old; he is the third child. Two children born previously were well-formed. He is healthy, but rather undersized. There is no family history

of congenital deformities. The birth was normal. The navel was prominent from the first, and became more so after the separation of the stump of the umbilical cord. The following notes were taken of the condition.

No umbilical scar is visible. The recti abdominis muscles are separated in their whole length, but most widely towards their lower part. The pubic rami are distant one inch from each other. Both testicles lie just outside the external abdominal rings. There is a poorly developed post-anal dimple. At the lower part of the abdominal wall, in the middle line and immediately above the root of the penis there is a distinct protrusion. The penis is upturned and lies closely applied to the under surface of this swelling. The projecting part is rather larger than a shilling-piece in circumference, and is surrounded by a distinct sulcus where it merges with the abdominal wall. This groove is deepest at the lower part of the protrusion. No signs of cicatrisation are present on its surface or margins. Its covering is white, but of a slightly pinker shade than that of the normal skin. It is thin, and when the child is at rest it shows numerous shallow folds. Measured in this condition, the swelling stands out $\frac{7}{16}$ths of an inch from the abdominal wall. The surface is dry. When the child cries a marked increase in size occurs; the protrusion is then $\frac{3}{4}$ths of an inch, while the covering becomes smooth and stretched and considerably darker in colour.

On gentle pressure the swelling can readily be reduced. The finger-tip passed backwards, invaginating the protrusion, can be felt to enter a distinct ring with sharp, thin edges. This ring allows the passage of the index-finger up to the middle of its terminal phalanx. On withdrawing the finger the swelling gradually reappears.

On several occasions out of many on which I reduced the swelling by pressure, a distinct gurgling sensation was transmitted to the fingers.

On bending down the penis from its abnormal position the following points are evident. From the lower part of

the lateral aspect of the protruded area there passes downwards on either side a thin red moist fold. These folds are $\frac{5}{16}$ths of an inch in length. They converge as they pass downwards, and blend together at the root of the dorsal surface of the penis. They enclose a triangular space with its base uppermost. The depth of this space is greatest at its base, where it measures $\frac{1}{4}$th of an inch; it becomes quite shallow towards its apex where the containing folds unite. This space is somewhat over-hung by its lateral folds passing outwards under them for $\frac{1}{8}$rd of an inch on either side. The floor of the space is moist and red; there is no escape of urine from it.

The lower sharp edge of the fibrous ring which limits the abdominal protrusion can be distinctly felt just on a level with the upper margin of this space, and pressure backwards on the floor of the space gives the impression to the finger of a thin fibrous membrane.

The penis is one inch long; it points upwards and forwards, at an angle of about 60° with the abdominal wall. It is slightly curved, with the concavity upwards. The dorsum of the body, and the proximal part of the glans lie closely applied to the under surface of the abdominal intrusion.

The only indication of the prepuce is a slight redundancy of skin passing from the frænum for $\frac{1}{4}$th of an inch on either side. Above this level, on the sides and dorsum of the penis the corona glandis is fully exposed, and the skin appears to be stretched presenting no folds.

The raphe on the under surface of the penis is well marked and passes back on to the scrotum.

The urethral orifice is normal in size and position, opening at the point of the glans slightly towards its under surface.

On the dorsum of the glans in the middle line there is a shallow groove running forwards from the corona. This groove is $\frac{1}{4}$th of an inch long and $\frac{1}{16}$th of an inch deep posteriorly. It becomes shallower anteriorly and ceases to be

evident $\frac{3}{16}$ths of an inch behind the external urinary meatus. Its surface is red and moist.

On the dorsum of the body of the penis another sulcus is visible. This starts towards the root of the organ, $\frac{1}{4}$th of an inch from its juncture with the abdominal wall, and passes forward directly in the middle line for $\frac{3}{8}$ths of an inch. It becomes shallower at its anterior exterior, which is $\frac{1}{4}$th of an inch behind the beginning of the groove on the glans. This sulcus is also deeper posteriorly where it is overhung by slight lateral and posterior folds, which give it, at this point, the appearance of the opening of a small sinus. The point of an ordinary probe is just admitted into this cul-de-sac. The lining of this groove is also red and moist. No escape of urine from either of these penile sulci has at any time been noticed.

Two lateral grooves, which do not meet in the middle line, are present at the junction of the penis with the abdominal wall. These are shallower than the sulci previously described. They begin abruptly towards the dorsum and gradually fade away as they pass round the sides of the penis.

The act of micturition is normally performed, all the urine being passed *per urethram*.

On two occasions I passed a soft catheter into the bladder, and drew off $1\frac{1}{2}$ ounces and 2 ounces of urine respectively. I then injected $2\frac{1}{2}$ ounces of sterile water, but this was insufficient to distend the abdominal protrusion. I did not think it wise to attempt to force in a greater quantity. A sound passed easily along the urethra; when in the bladder, by depressing the handle, the beak of the sound slipped readily into the protruded portion and could be made to project its covering. The thickness of tissue between the sound and the fingers palpating it in this position was certainly not more than $\frac{1}{8}$th of an inch.

On withdrawing the sound from the protrusion and passing it well up towards the fundus of the bladder, the instrument was felt through the abdominal wall one inch above the projecting area. On firmly pressing the sound

upwards in this position, the thin coverings of the protrusion became puckered inwards, and the whole swelling was flattened out.

I have a photograph which shows the projection from the abdominal wall, the relative position of the penis to it, the absence of the umbilicus in the normal position, and the incompletely descended testicles.

FIG. 8.

A radiograph taken by Dr. Holland shows separation of the pubic rami. An oval shadow is present, giving an appearance similar to that produced by a vesical calculus. No stone, however, was detected in the bladder. This shadow corresponds with the position of the abdominal protrusion and upturned penis, but I fail to see how these can possibly account for it. Rectal examination revealed nothing. We had proposed taking another radiograph, but the child developed measles, and had to be sent out of the hospital. For the same reason I was unable to obtain a cast of the parts.

My view is that the abdominal protrusion is a closed and dilated urachus. That when the child cries the pos-

terior wall of the bladder is forced forwards into it, and that under extra strain intestine also finds its way in behind the bladder, as evidenced by the occasional gurgling sensation felt on reduction.

That the protrusion occurs at the point where the umbilical cord was originally attached is, I think, apparent from the history, from the absence of an umbilical scar higher up, and from the distinct fibrous ring which can be felt on inverting the protrusion.

The bladder has obviously developed upwards, beyond the level of the abdominal projection as ascertained with the sound. That the anterior wall of the bladder is attached to the lining of the protrusion is proved by the inward puckering of the latter, when the sound is pressed upwards against the fundus.

The case is of interest from a developmental standpoint, and also because I find no similar condition reported in the literature of the subject.

Kermission,* in discussing extrophy of the bladder, gives the following classification.

1. Ectopia of the bladder without division of its walls.
2. Superior vesical fissure.
3. Inferior vesical fissure.
4. Complete extrophy.

He cites one case reported by Froriep and Gusserow as the only known example of the first variety.

In this case there was a hernia of the bladder through a patent urachus. Urine escaped partly by the abdominal opening, partly by the urethra. The posterior bladder wall was everted through the urachus when the child cried, bringing the ureter orifices into view. The umbilical scar, penis and symphysis pubis were normal. I briefly mention this case on account of its relation to the present one.

In considering the developmental effects which are present in my case, I look upon a dilated allantois with stunted development of its stalk, and consequent short

* 'Traité des Maladies Chirurgicales d'origine congénitale,' 1898.

attachment to the chorion as the probable factor in producing the deformity. A constant pull downwards would arise from such a condition, and thus account for the low position of the umbilicus corresponding to that found in an early fœtal state. This traction, associated with the dilation of the allantois, would form a mechanical obstruction to the development of the abdominal wall. It would account for the large size of the umbilical ring, the thin covering of the abdominal protrusion, the failure of coalescence of the cutaneous folds below it, and the non-union of the pubic bones.

The theory advanced by Ahlfeld,* that an abnormal position of the umbilical vesicle—its direction being towards the caudal rather than the cephalic end of the embryo—may cause traction on the omphalo-mesenteric canal, and as a result non-closure of the abdominal wall, might also account for the allantois protrusion, and possibly the non-development of the lower abdominal wall and pubic bones in the present case.

But, I think that a persistent early condition of the allantois, such as I have described, is more likely to have been the causal factor.

In Gusserow's case a dilated urachus was apparently laid open during the separation of the cord, and it is easy to see how such a complication might have occurred in the present one. Had this taken place a similar protrusion of uncovered posterior bladder wall must have followed.

In Gusserow's case the development of a normal stalk to the allantois, and the consequent absence of downward traction and mechanical interference with development would explain the normal position of the umbilicus, the complete abdominal wall, and united symphysis pubis.

In regard to the development of the parts involved, it is now agreed that the anterior wall of the bladder, and the lower part of the anterior abdominal wall have a common origin. They are derived from the so-called anal membrane, the most posterior reflected part of the primitive

* Ahlfeld, 'Die Missbildungen des Menschen,' t. ii, p. 205.

streak. Arrested development of this anal membrane occurring at an early period would thus lead to deficiency in both bladder and abdominal wall, such as exists in complete extroversion.

But, just as we meet with various degrees of imperfect closure of the branchial clefts, I see no reason why we should not have intermediate stages between complete extroversion of the bladder and the normal.

In the case in question, the mesoblastic elements comprising the anal membrane, although stunted in development as evidenced by the separation of the recti and non-union of the pubic bones, have still united in the middle line below the abdominal protrusion in the form of a thin fibrous membrane. Development of the epiblastic structures has not advanced to the same degree, with the resulting formation of the ununited cutaneous folds which are present at the lower part of the abdominal wall.

Should the urachus have been opened in the separation of the cord, and had the mesoblast only developed to the extent of the epiblast there would, I think, have resulted a deficiency of both bladder and abdominal wall, forming a complete vesical extrophy.

In regard to the grooving on the dorsum of the penis, and the imperfect development of the upper part of the prepuce, I believe that we can most reasonably account for it as being a continuation downwards of the imperfect development of epiblastic elements. In other words, a failure in complete union of the most superior portion of the folds of Rathke. Here again, had the mesoblast also remained undeveloped complete epispadias would have resulted. Such a view is, I consider, more tenable than that complete torsion of the penis has occurred, as there is no evidence of such twisting, and the meatus externus and urethra are normally placed towards the lower part of the penis.

The lateral grooves at the root of the penis may, I think, be regarded as resulting from the upturned position of the organ. (*Read November the* 21*st*, 1902.)

12. A CASE OF LEUKÆMIA IN A BOY AGED 3½ YEARS, ASSOCIATED WITH TUBERCULOUS MESENTERIC GLANDS.

By GEORGE CARPENTER, M.D.

Leonard P—, aged 3½ years, was admitted into the Evelina Hospital on July the 7th, 1901. He had been ill for five weeks, and his pallor on admission was extreme. Tuberculosis was stated to be present on the maternal side of the family, and there had been instances of it on the paternal. His father, so it was stated, had always been pale, and his two paternal aunts suffered from anæmia badly. There was no history of malaria or syphilis. The child was healthy at its birth, it was fed on the breast for nine or ten months, and passed through an uneventful infancy. There was nothing worthy of note in its dietetic history, and its hygienic environment was always quite satisfactory. Five weeks before being brought to the Evelina Hospital his mother noticed that he suddenly, as she states, became quite pallid; but his appetite at this time was fairly good. A fortnight later he developed a croupy cough; then he became languid and would not take his food. A doctor now saw him and pronounced him to be anæmic; he was treated for this for a short time, and was then brought to the Evelina Hospital. During this latter period of his illness he lost flesh and was thirsty, and for three days prior to his admission into the hospital he passed through an attack of sickness. His abdomen also became painful, but only sufficiently so to make him cry on one occasion. He was also at this time so short of breath that he could not walk.

On examination he was found to be a very pallid child with white lips, pale gums, and a complexion of a lemon-yellow colour. He was dark and puffy-looking under the eyes, the sclerotics of which were bright and glistening.

There were some bruises on his trunk and limbs. He was very restless, constantly shifted his position in his cot, and flung his arms and legs about. His abdomen was distended. The free edge of the spleen could be felt from two to three fingers' breadths below the costal margin, and the liver free edge was felt to a similar extent below the costal margin in the nipple line. The accessible lymphatic glands were not enlarged. The heart showed some epigastric pulsation, and there was a systolic hæmic bruit, which was best heard over the left fourth and fifth intercostal spaces. The second pulmonary sound was loud. There was a trace of albumen in the urine on his admission, but this was only observed on that solitary occasion. His temperature was 99·4° F., the pulse-rate was 128 to the minute, and the respiration-rate 28 to the minute.

The blood examination was as follows. Red corpuscles, 1,568,111 per c.mm.; hæmoglobin, 15 per cent.; colour index, 0·5; white corpuscles, 130,111 per c.mm., the ratio being 1 to 12. Fresh films showed an increase of the white corpuscles, and the red were in rouleaux. There were no microcytes or poikilocytes. Stained films showed an increase of leucocytes, mainly small lymphocytes. There were a few polymorphonuclear leucocytes and myelocytes, and some doubtful nucleated red corpuscles (megaloblasts). During his short stay in the hospital his temperature was somewhat above normal, the highest point registered being 100·2° F. His respiration-rate varied between 28 and 44 to the minute, and his pulse-rate between 126 and 152 to the minute. He was sick on one occasion, and once he complained of pain in the left side. On the evening of the 14th he became collapsed, and made frequent complaints of pain in his abdomen. At 9.30 p.m. he commenced to scream and throw his arms about, and was noticed to be squinting. After a dose of morphia he became quieter and slept until 2 a.m., at which time he was very thirsty and constantly craved for drink; subsequently his head was bathed with profuse perspirations, and he started screaming again at 3.30 a.m., and then his

pulse was barely perceptible at the wrist, and his respirations were gasping. He died somewhat later.

The *post-mortem examination* was made by Dr. Nabarro eleven hours after death. The body nourishment was good, and there were no signs of decomposition. The body was extremely pale, and there were several purple-coloured bruises on the leg and one over each frontal eminence. The nervous system was not examined by request. There were petechial hæmorrhages all over the surface of the heart; it contained pale clots and clear serum. The valves were healthy. The cavity of the left ventricle was dilated. The heart muscle was very soft and pale, and "tabby-cat" striation was slightly marked in the papillary muscles. The blood throughout the body was pale. The bronchial glands were enlarged and very dark, as though some hæmorrhage had occurred into their substance. There were several ounces of clear fluid in each pleural cavity, and subpleural ecchymoses were present on the surface of both lungs. On section the lungs were somewhat œdematous. There were no clots in the pulmonary artery or its branches. The stomach showed some submucous hæmorrhages. The intestines displayed unduly prominent Peyer's patches and lymph-follicles. The jejunum appeared to be thinner than normal. The lymphatic tissue in the large intestine, as in the small, was in excess, and a few small ulcers were present in the large and small intestines. The mesenteric glands were very considerably enlarged; they were pale in colour and the largest of them were caseating in the centre. The liver was large and very pale. The spleen was large, it weighed 4 ounces and was normal in appearance. The supra-renal bodies were normal. Kidneys.—The right kidney was very large and weighed 5¼th ounces; its surface was covered with ecchymoses, and its capsule was not adherent. On section the cortex was greatly thickened. The left kidney was similar, but larger; it weighed 6¼th ounces, and at its upper part was flattened by pressure from the spleen. The pancreas was normal.

Lymphatic glands.—Many small glands were present along the course of the abdominal aorta and iliac vessels, and also in the groins and the axillæ. They were caseating. The thymus was apparently normal.

A piece of rib was removed; on section the marrow was pale, but apparently normal. Cultivations were taken from the right ventricles, but yielded negative results. Microscopical examination of the thickened renal cortices showed extensive leucocytal infiltration.

(*Exhibited November the 21st*, 1902.)

13. TUBERCULOSIS OF THE CONJUNCTIVA.

By SYDNEY STEPHENSON, C.M.

Mabel H—, aged 4 years, was brought to the eye department of the North-Eastern Hospital for Children on November the 12th, 1902, on account of a drooping of the upper lid of the left eye and a swelling of the corresponding side of the face.

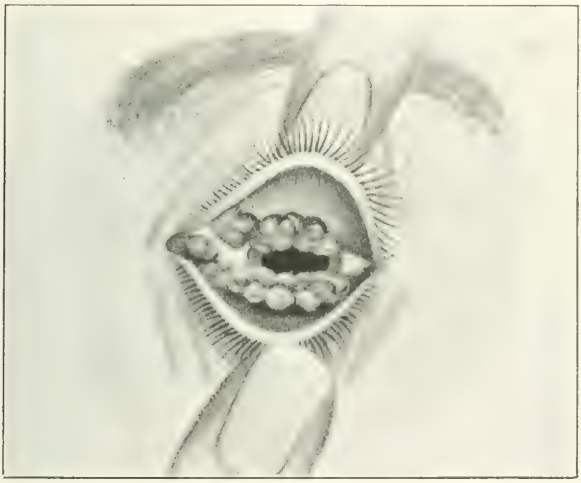
Family history.—There is said to be no "consumption" on either side of the family. The patient is one of seven children of whom five are living. One child died, aged 6½ years, from "inflammation of the kidneys;" and the other, aged 13 months, from "measles and cold, and inflammation of the lungs."

Personal history.—She had measles eighteen months ago, immediately followed by whooping-cough. Two months ago (September, 1902) it was noticed that the glands below the left lower jaw were enlarged, and that the left eye looked heavy, was red, and discharged "matter." Since these symptoms were observed, the child has lost flesh.

Present condition.—She is a very pallid-looking child,

weighing (with clothes) 38 lbs. There is a marked swelling of the left side of the face and neck, due partly to induration and swelling of the preauricular and submental glands, and partly to a diffuse lymphangitis of that side of the face. To be more precise: below the centre of the left half of the inferior maxillary bone is an enlarged gland measuring about one inch by half an inch. It is free from tenderness, and does not appear to be undergoing any degenerative changes. There is another gland about half an inch in diameter, circular in shape

Fig. 9.



and freely movable, situated in front of the left sterno-mastoid muscle, about one inch below the gland mentioned above. The left preauricular gland is enlarged. The posterior cervical and the inguinal glands upon both sides are slightly enlarged. There is commencing caries of the first molar tooth on the left side of the lower jaw. No glandular enlargements can be felt in the axillæ. The tonsils are swollen but not inflamed. No abnormal physical signs can be detected in the lungs. Neither the liver nor the spleen appears to be enlarged. There is no lupus,

either of the skin or mucous membrane. There are no tubercles in the choroid. The tongue is slightly furred. The urine is acid, and contains urates, but neither albumen nor sugar. Left eye: the lids are semi-closed and obviously thickened, probably, like the side of the face, from lymphangitis. There is a considerable amount of trachoma-like material in connection with the semi-lunar fold. On everting the eyelids, the lower palpebral conjunctiva is found to be bestrewn with miliary granulations and folds of cockscomb-like tissue (see Fig. 9), but no ulcerations can be found. Similar changes affect the superior retro-tarsal folds. The tarsal conjunctiva of the upper lid, on the other hand, can scarcely be said to be involved by the morbid process. The cornea is clear. Right eye: this is unaffected.

Diagnosis.—Some of the more prominent granulations were excised from the left lower eyelid, hardened in rectified spirit, embedded in celloidin, and cut upon a Leitz's microtome. The sections when examined microscopically showed a typical picture of tuberculosis, the giant-cell systems being especially well marked. A restricted number of tubercle bacilli were found, but only after many specimens had been examined.

(*Exhibited November the* 21*st,* 1902.)

Discussion.

Mr. A. H. Tubby said he had, at the present time, a particular interest in a case showing a tuberculous patch in the choroid. Three years ago he was asked to see a child with some doubtful hip-joint trouble, and he took a rather serious view of the case. Another surgeon was called in, who seemed inclined to take rather an optimistic view, thinking the condition would settle down quietly. He (Mr. Tubby) persevered in his opinion, however, and in a very curious manner the diagnosis had been confirmed. After he had allowed the patient to walk about he received a letter saying he had been taken to an ophthalmic surgeon, who said there was a curious patch occupying the yellow spot and the area around it, and he wanted to know whether he (Mr. Tubby) had considered the hip tuberculous.

He had entertained no doubt about the matter, and the choroiditis made clear the nature of the case.*

Postscript.

The anterior chamber of a rabbit's eye was inoculated with a morsel of diseased tissue taken from the patient's lower lid. In five weeks' time there was tuberculosis of the iris, ciliary body, and cornea, with a tendency to prolapsus iridis. The animal was killed on January the 7th, 1903. Tubercle bacilli were found in abundance in exudation taken from the anterior chamber, smeared upon cover glasses, and stained by the Ziehl-Neelsen method. The changes characteristic of tuberculosis were found with the microscope in sections of the iris and neighbouring parts. Tubercle bacilli were further demonstrated in those structures.

Progress and treatment.—Meanwhile the affected conjunctiva was treated by exposure to the X-rays at a distance of six to ten inches from the focus-tube, for an average period of ten minutes at each sitting. Between December the 2nd, 1902, and January the 1st, 1903, nine such exposures were made, and the local condition underwent a great improvement. Beyond bathing the eye three times a day with 1 in 5000 sublimate lotion, and giving cod-liver oil and steel wine internally, no other treatment except the X-rays was adopted.

Owing to an attack of varicella, the child was discharged from the hospital on January the 7th, 1903. It may be noted, in passing, that the temperature during her detention had risen once only to 99° F.

The patient was readmitted on January the 20th, 1903. The conjunctival condition was practically cured. The enlarged glands below the jaw and in the neck, however, had become larger and adherent to one another, and so an operation for their removal was undertaken on

* "Tuberculosis of the Choroid," by George Carpenter, M.D., and Sydney Stephenson, C.M., 'Reports of The Society for the Study of Disease in Children,' vol. i, pp. 169—181.

February the 6th, 1903, by a surgical colleague, Mr. Henry J. Curtis. Two separate incisions were made—one below the jaw, and the other in the neck. One of the cervical glands was adherent to the internal jugular vein, and had to be carefully separated from the latter vessel. One of the glands removed by Mr. Curtis was cut open, and several cover-slips lightly smeared with the cut surface. These, when stained by the Ziehl-Neelsen method, showed scattered tubercle bacilli. Another gland, examined with the microscope, was found to be caseating, and sections stained with carbol gentian violet and counterstained with vesuvine showed tubercle bacilli.

The left eye was exposed on four more occasions (February the 21st and 28th, March the 5th and 10th) to the X-rays, the eye being distant six inches from the focus-tube, and each exposure lasting ten minutes.*

On April the 3rd, 1903, the general condition of the child was excellent. The left eye appeared to be wholly free from disease, and the only cicatrices present in the conjunctiva were those produced by the removal for diagnostic purposes of diseased material.

In conclusion, I may add that, so far as I know, this is the first instance upon record where tuberculosis of the conjunctiva has been cured by the X-rays.

14. TUBERCULOUS PERITONITIS: ETIOLOGY.

By EDMUND CAUTLEY, M.D.

On the grounds of scientific precision all forms of inflammation of the peritoneum due to the tubercle bacillus should be included under the name of "Tuberculous Peritonitis."

* I have to thank Mr. A. E. Pinniger, of the North-Eastern Hospital for Children, for having carried out the treatment.

Preference is sometimes given to the name "Tuberculosis of the Peritoneum," for peritonitis suggests that the essential feature is an inflammation, whereas the tuberculous infection is the great characteristic. Both the clinical signs and symptoms are rarely those which we are accustomed to associate with a diagnosis of peritonitis.

In one variety, the most common one in children, the peritoneum may be studded with grey tubercles without the spread of inflammatory changes from the tubercles to the peritoneum. This we may omit from the discussion as being part of a general blood infection, an acute miliary tuberculosis. Death usually takes place before the onset of any definite abdominal symptoms. Should the illness be more prolonged the grey tubercles may undergo caseous degeneration, and there may result a certain amount of peritonitis and exudation. Strictly speaking, the process, even in the earliest stages, is an inflammatory one, for a tubercle results from inflammatory changes initiated by the irritation of the tubercle bacillus. Omitting this variety we have three more or less distinctive types of the disease, any one of which may be either localised or generalised. This classification I suggest as the most simple one for the purposes of a discussion :

1. Adhesive peritonitis.
2. Peritonitis with effusion of (*a*) serum, (*b*) pus.
3. Caseous or ulcerative peritonitis.

From the etiological point of view no such distinction is necessary, and my remarks will apply to every form of tuberculous infection of the peritoneum. The etiology of the disease is that of tuberculosis generally, with the addition of a few special points indicating why the peritoneum is attacked.

Gentlemen, the offspring is the product of inheritance modified by environment. In the case of disease due to microbial infection we have the same idea expressed in another form, when we speak of "the seed and the soil." The latter phrase does not quite cover the ground, for it omits special reference to the manures modifying the nature of the soil.

By the *soil* I mean the primary physical constitutional state of the subject dependent on inheritance.

By the *manures* I mean all those influences or modifications which affect the nature of the soil, the constitutional state, as a result of environment.

The *seed* is the tubercle bacillus, for that, and that only, can give rise to tuberculous disease.

Inheritance.—Like many of the beliefs of our ancestors, the belief in the inheritance of tuberculosis has been scoffed at and deemed of no account. On the other hand, it has undoubtedly received undue prominence as an etiological factor, and has caused much too gloomy a prognosis to be given in the case of children of tuberculous parentage. The term inheritance in this connection means one of two things.

1. Either the disease is directly transmitted from parent to child through the medium of an infected germ cell, or maternal blood supply.

2. Or the child inherits a certain peculiarity or delicacy of structure, which renders it more susceptible to the particular disease.

Vague and indeterminate as the latter explanation sounds, it is probably true of the inheritance of disease. The physical strength and health of the child, and its power to resist disease, depend primarily on those of its ancestors. Whether the child of tuberculous ancestry is or is not more prone to develop tuberculosis is a moot point, which will be referred to subsequently.

Taking the case of the father first, it has been shown that the tubercle bacillus may exist in the semen, *e. g.* in tuberculous disease of the vesiculæ seminales. It is almost inconceivable that the microscopic spermatozoon can actually contain the bacillus, even in spore form, and convey it to the ovum. Much more probably the infection of a sperm cell would impair its nutrition and render it useless for the purpose of impregnation.

Similarly, it is equally improbable that an infected ovum could be satisfactorily impregnated and carry out

developmental processes. The bacillus has been actually found in the ovum in one instance by Baumgärten, who artificially fecundated a rabbit with tuberculous semen. But maternal inheritance stands upon a very different footing. For nine months or so of pre-natal existence the future child is in the closest of all possible contact with the mother. During the whole of this period it receives its blood supply from the mother, filtered, it is true, for the greater part of the time through the placenta. Yet even human filters are sometimes imperfect. Supposing the mother to be tuberculous, it is by no means improbable that the placenta may become affected. Its nutrition may be sufficiently impaired to permit the passage of the bacillus from the maternal blood to that of the fœtus. Or the placenta may be actually tuberculous. Gärtner, as the result of inoculating the mother, intra-peritoneally or intra-venously, produced tuberculosis in young mice. A calf has been found tuberculous at birth. Bacilli have even been found in the human fœtus removed by Cæsarean section from a tuberculous mother. Many infants show such advanced tuberculosis of the lungs (and note that it is usually of the lungs) a few months after birth as to render fœtal infection a suggestive explanation. On the other hand, in most of these instances the mother has shown no evidence of tuberculosis, and there has often been a history of exposure to infection. Even if there be no tuberculosis in the mother, as in the case of a child of six months under my care, in whom extensive tuberculosis of the lungs was found, the probability of direct infection is enormous. In a case of Stiegenberger's, a babe died at five months from extensive tuberculosis. The parents were healthy, but the babe had been nursed by a tuberculous nurse.

In the bulk of supposed inherited cases of tuberculosis we shall find on careful investigation evidence of direct infection, rather than of infection through the medium of a germ cell or maternal blood supply. Inheritance of the disease is so rare as to be a negligible quantity, and the

spread from parent to child is in very truth by direct infection.

The soil.—On the primary constitutional state depends to a certain extent the susceptibility to tuberculosis. On certain unknown bio-chemical characteristics of the tissues depends the growth of the bacillus, when once it has obtained an entrance. Of the latter conditions little is known beyond the fact that the bacillus does not grow in an acid medium. Consequently it is rare to find tuberculous disease of the stomach.

It is a moot question whether the children of a tuberculous parent are more liable than others to contract tuberculosis, apart from the risk of direct infection. Evidence has been brought forward by Squire and Maxon King in favour of the view that there is no such predisposition. It is sometimes even supposed that there is an acquired natural immunity, and in favour of this it must be borne in mind that such children are much more exposed to infection than others. The case incidence should be, therefore, much higher. According to Maxon King it is actually lower.

The resistance to infection is only a question of degree, and is largely influenced, increased or decreased, by surrounding conditions. Probably all animals can be infected under suitable conditions as to dosage and surroundings. What is true of animals in this connection almost certainly applies equally to children.

The seed.—According to Möller there are as many as nine acid-resisting bacilli which stain like the tubercle bacillus. Notable among these are the butter bacillus and the smegma bacillus. Care must be taken not to rely upon staining properties as proof that a suspicious bacillus is the tubercle bacillus. The most certain proof is that obtained by the intra-peritoneal inoculation of guinea-pigs.

From the moment of birth the child is liable to infection from many sources. Now, I do not suffer from bacillophobia, but if there were no tubercle bacillus there would be no tuberculosis. Yet the bacillus need not be the

terrible enemy some of us consider it. Granted that its ravages are great, it does not follow that we are going the best way to eradicate the disease by building sanatoria to keep alive those already infected, enabling them to propagate their species, even in sanatoria; or by passing bye-laws to prevent the homely habit of spitting, or the custom of kissing those who are not clean-shaven. Surely this is not the ideal way of attacking a widely prevalent disease. The farmer who wishes to keep thistles out of his land does not set up wind-screens to keep out the thistle down. Such an attempt would be as foolish as it would be unavailing. We can, perhaps, diminish to a slight extent the distribution of the tubercle bacillus, but, like the farmer, we must devote our energies mainly to tilling the soil effectually, to increasing the resisting powers of the individual until he can scoff, as many a healthy man now scoffs, at that arch-fiend the tubercle bacillus. If preventable, why not prevented? The man in the street is governed by phrases, and this has caught his ear. How are we to answer him? Gentlemen, the future of the nation is in the hands of little children. The future of the nation is in the hands of our profession, for on us depends the health of the children. Let us increase their powers of resistance to infection and diminish the liability to exposure. If the constitution is sound and the environment as perfect as can be obtained in this imperfect world, then the bacillus becomes a comparatively harmless foe.

The environment, or manures.—On the other hand, the strength of the foe is increased by every factor which lowers the health of the child. If in addition there be inherited delicacy or weakness of constitution due to parental tuberculosis, syphilis, alcoholism, or mere delicacy, the danger of bad environment is exaggerated. Everything which lowers the health of the child, such as impure air, lack of sunlight, close rooms, unsuitable or insufficient food, imperfect or badly applied clothing, and bad hygienic conditions generally, acts as a predisposing cause of tuber-

culous disease. So, too, local or general debility of the tissues may be due to local or general disease or injury and, by lowering the vital resistance, predispose to infection.

Under all these conditions, the ubiquitous bacillus may find on entrance a suitable soil for its growth and development in the damaged area of impaired vitality.

But though the seed may enter, and does enter, it does not always acquire a holding and flourish. Sometimes it falls on stony ground. The soil is not always suitable. Thus, clinical evidence favours the view that the uric acid diathesis is resistent to tuberculous infection.

Mode of infection.—The bacillus enters by the respiratory tract, the alimentary tract, or the cutaneous system. Infection through the skin is rare in adults and still more rare in children. We need not consider it. The aërial convection of tuberculosis has been proved by direct experiment in animals, by diffusing dry powdered sputum in the hutches of rabbits. Probably this is the great source of infection in man. The possibility of such infection is great. It has been estimated by Böllinger that one cubic centimetre of sputum may contain a million bacilli. According to Nuttall, as many as 4,000,000,000 may be expectorated in the space of twenty-four hours. Fortunately most of them are dead bacilli. Still, it is clear that the neighbourhood of a consumptive, who promiscuously spits about the floor, may be highly infectious. In coughing or speaking loudly bacilli may be mingled with the spray. If the patient uses handkerchiefs these, too, may convey the bacillus, and it may be spread by direct contact with the lips, etc. After puberty, the lungs are almost invariably found affected in cases of tuberculosis. In childhood we also find that either the lungs or some of the thoracic glands are almost always affected. In a paper read before this Society in November of last year, on "The Etiology and Morbid Anatomy of Tuberculous Meningitis,"*

* 'Reports of The Society for the Study of Disease in Children,' vol. ii. pp. 53—66. "Observations on the Etiology and Morbid Anatomy of Tuberculous Meningitis," by Edmund Cautley, M.D.

I brought forward evidence to show that the lungs or bronchial glands, or both, were almost invariably infected. In no less than twenty-three out of twenty-five cases, fully examined post mortem, some one or more of the mediastinal glands were caseous. In the other two none of the glands appeared affected. In four the mesenteric glands were also involved. This evidence I took to be opposed to the view that infection takes place through the alimenta. I expect that pathological evidence will be in favour of a similar proposition in the cases of tuberculous peritonitis. It is rare for the disease to be limited to the abdominal cavity. On the other hand, infection through the lungs is not the simple matter we might regard it. Considering the laws of diffusion of gases, the filtering of air through the nose, the ciliary action of the cells lining the trachea, the results of experiments with the inhalation of heavy dust, and the differences in the localisation of the disease in the lungs of adults and children, it might be fairly urged that the bacilli do not reach the lungs through the inspired air, but through some other channel, such as the mucous membrane of the mouth, tonsils, pharynx, etc., *viâ* the lymphatics and glands of the neck.

Many regard the alimentary tract as the chief channel of entrance of the bacillus. This brings us to the question of tuberculous milk. That there is a good deal in the market is undoubted, though I think the prevalence of tuberculosis in cattle and of tuberculous disease of the udder is exaggerated. It is stated that no less than 30 per cent. of all cows are tuberculous, and that 3 per cent. have tuberculous disease of the udder. I do not believe it, and it is opposed to my experience. Opposed also to the view that tuberculous milk is the great source of infection, I should like to quote Professor Carlo Ruata, of Perugia, who maintains that the milk of tuberculous cows never produces tuberculosis in man; that in adult life primary intestinal tuberculosis does not occur; and that in the first years of life tabes mesenterica, as well as other forms of tuberculosis, is always inherited.

Not many will, I think, support this statement in its entirety.

The question of the identity of the human with the bovine bacillus is still a matter of uncertainty, though on the whole the probabilities are strongly in favour of identity, modified by environment.

To the professorial mind of Koch a few experiments with negative results were sufficient justification for stating that the bacilli are different; that the human bacillus will not give rise to bovine tuberculosis; and that the bovine bacillus will not cause tuberculosis in the human subject. From this he argued that tuberculous milk is harmless; and he supported his views by asserting the infrequency of primary intestinal tuberculosis in infancy, and by the absence of wide-spread epidemics due to the milk of tuberculous cows. It is assumed that alimenta can only produce the disease by setting up a primary tuberculosis of the intestine. That such disease is rare is shown by the results of autopsies on tuberculous children. Biedert found primary intestinal disease in only 16 out of 3104 cases. Baginsky found in 933 cases no such disease, unless the lungs and bronchial glands were also affected.

Many observers have succeeded (Böllinger, Crookshank, Sidney Martin, de Jong, Arloing, Ravenal, Wolff) in showing that the human bacillus will produce tuberculosis in cattle, chiefly by experiments on calves, although it has not the same degree of virulence as the bovine bacillus. In fact, the bovine bacillus appears to be more virulent to all animals used for experimental inoculation. Probably Koch's negative results of experimental inoculation were due to using too small doses of insufficient virulence.

Another argument against Koch's views is that it can be shown, by feeding experiments, that tuberculosis may result without the production of a primary lesion. Thus, the cervical glands of a pig (Woodhead) and the mesenteric glands of the guinea-pig (Cornil and Babes, Baumgärten, Klebs, etc.) may become tuberculous, although no local evidence of disease can be found. Bacilli have been found

in the tonsils with an apparently intact mucous membrane (Dieulafoy, A. Latham). The experimental infection of the nasal mucosa by rubbing on sputa will cause tuberculosis but no local lesion (Cornet, Renshaw). Thus it appears that the bacillus can enter through an intact mucous membrane, *i. e.* intact to the naked eye. Whether it can pass through a healthy mucosa is a matter of doubt. It is so easy to suppose some local defect of nutrition or vitality to account for it.

Milk is the staple article of diet during the first year or more of life. In the case of hand-fed children it is usually cows' milk, especially among the better classes. There is no doubt that much milk is sold containing the tubercle bacillus. Yet tuberculous lesions in the abdomen are not very common. They are more common among the poor than the rich, although, or perhaps because, the former get less milk. In addition, the milk obtained by the poor is likely to be poorer in quality, cheaper, and perhaps infected, whereas among the better classes more care is taken in obtaining and preparing it. Among the poor all the predisposing factors are much more in evidence, but I can find no indication that a higher percentage of children fed on cows' milk get tuberculous peritonitis than of those fed on condensed milk. I regard the latter as the more susceptible.

The alimentary canal is certainly very insusceptible to infection by the human tubercle bacillus. This is shown by the frequency with which we find no intestinal infection and no caseation of mesenteric glands in infants and young children who have been for weeks or months swallowing sputa from tuberculous cavities in the lungs. Still, as the result of autopsies on children at Great Ormond Street Children's Hospital, states that 59 per cent. of tuberculous children have tuberculous mesenteric glands, and that 80 per cent. of cases of tuberculous mesenteric glands showed ulceration of the bowel. From this it follows that 47·2 per cent. of tuberculous children have ulceration of the bowel, a percentage which seems unduly high.

Statistics like this strongly suggest alimentary infection. We ought, indeed, to have large numbers of instances of alimentary infection if tuberculous milk is widely distributed, and if the bovine bacillus is much more virulent than the human variety. There is no reason for assuming that man is an exception to other animals in the degree of susceptibility to infection by the bovine bacillus. Supposing the bacillus to pass through the stomach uninjured, and to be able to pass through the intact mucous membrane of the intestinal tract, two channels are open to it. It may pass into the blood-stream and be carried to the liver. If it take this course the liver must have a powerfully destructive effect, for tuberculous foci in the liver are rare, except in the case of an acute general dissemination. Or it may pass by the lymph channels to the glands and be there destroyed, or set up caseation. The glands act as a dam, and should the dam fail or burst, the bacilli may pass onwards into the thoracic duct and so to the blood, giving rise to general infection.

Assuming that the bacillus reaches the systemic circulation by this course, why is it that the lungs and thoracic glands are the organs commonly infected? The infected blood will be distributed throughout the systemic circulation and, with the exception of that which passes by the bronchial arteries, will only reach the lungs after passing through the body and the right side of the heart. The bronchial glands are caseous in 75 to 80 per cent. of tuberculous children. Not all the lymph from the intestines passes through the glands, so it is possible that the lymph-stream may be infected and the bacillus may set up tuberculous disease in glands further on, such as the mediastinal, or may pass into the thoracic duct and blood-stream without going through a gland.

We have therefore to assume that if the bacillus reaches the bronchial glands *viâ* the intestinal canal it does so by evading the mesenteric glands or by passing into the blood-stream, and then for some cause or other being carried to these glands. Much more evidence is required

before we shall be justified in accepting such an assumption.

Tubercles may occur in and block small arteries or veins. They may break down and cause general dissemination. Such an explanation might be theoretically true of some cases of tuberculous peritonitis. It is not a wild speculation to suppose that the tuberculous process might extend from a gland to an artery, or that an artery might be directly infected through its vasa brevia, and that on breaking down a dissemination in the parts supplied by that artery might result. So far I have been unable to find clear post-mortem evidence of such limited arterial distribution.

Wide dissemination suggests distribution of the infective agent by the blood-stream. Wide dissemination is common in infancy, and is most commonly secondary to caseation of a thoracic gland. Dissemination by the blood-stream does not, however, clearly explain the limitation of the spread to the peritoneum. We are driven to the view, that if this be the mode of infection, the bacilli must be simultaneously and equally widely distributed throughout the systemic circulation, but that they are destroyed elsewhere than in the peritoneum. We may note in this connection how rarely tuberculous nodules are found in the skin, and that in acute miliary tuberculosis few tubercles are found except in the lungs, unless the illness is rather prolonged. It is probable that in the healthy peritoneum, as in other tissues, the bacilli are destroyed, but that if the resisting power of this large serous membrane be impaired a tuberculous peritonitis will ensue.

As a paralled to this I would suggest that outbreaks of miliary tuberculosis can occur in the system as a whole, or in portions of it, without causing the death of the child. Just as we get ocular evidence of the recovery of cases in which the peritoneum has been found studded with tubercles, so we have clinical evidence which indicates to me that some of the cases diagnosed as capillary bron-

chitis, ending in recovery, may have been due to an attack of dissemination of miliary tubercle in the lungs.

Though I am inclined to the view that tuberculous peritonitis is usually due to infection through the blood-stream, I regard the caseous form as more commonly due to rupture of a caseous mesenteric gland, or to extension from such a gland, in a somewhat similar manner to the extension from a caseous bronchial gland to the lung. At present we seem hardly justified in stating that the mesenteric glands become caseous as a result of infection from the intestine. It is quite possible that the bacillus is carried to the gland by the blood. Even in advanced cases of tuberculous ulceration of the small and large intestine we do not invariably find caseous mesenteric glands. This was very remarkable in a case I showed before the Medical Society.* The ulceration was extreme, the glands were not caseous, the peritonitis was limited to the bases of the ulcers, and there was an old tuberculous focus in the apex of one lung. If we get no caseation in the glands in such a case as this, how much less probable is infection of the glands through the mucous membrane of the healthy intestine. In a few instances the peritonitis appears to be secondary to a primary tuberculosis of the genital organs. More often both are secondary.

The predisposing causes which render the peritoneum susceptible are mainly those due to improper feeding, gastro-intestinal disturbance, and general malnutrition. Another factor, very prevalent among the poor, is unsuitable clothing. We constantly see that the ordinary child of the poor is badly clad, with the clothes so arranged as to hang from the shoulders. When the child is lifted up, under the arms as so often happens, the abdomen is seen to be bare or, at any rate, only protected by a thick layer of dirt. In young children, and in girls for many years, the abdomen is insufficiently protected. Perhaps this is the explanation of the greater frequency of tuberculous peritonitis in females in later life.

* 'Med. Soc. Trans.,' vol. xxiv, p. 272.

Constantly in the early stages of the disease we find a distended belly, flatulence and constipation; a condition we cannot invariably diagnose as incipient tuberculous peritonitis. Such symptoms often, but not invariably, prove tuberculous.

In conclusion, I would suggest that it is not proved that all forms of tuberculous peritonitis are due to the same brand of bacillus. Both clinically and pathologically we seem to have grounds for supposing that the bacillus exerts a different effect, according as it is of the human or possibly the bovine species. Certainly, in some instances of chronic tuberculosis in children the character of the spleen closely simulates that seen in the tuberculosis of guinea-pigs. The question is one well worthy of patient elucidation.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902.)

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15. TUBERCULOUS PERITONITIS: SYMPTOMS AND DIAGNOSIS.

By JAMES BARR, M.D.

The task set me of opening that part of the discussion dealing with the symptoms and diagnosis of tuberculous peritonitis is a very important one, and I should like to give it a much more definite and prominent position than that which it occupies in text-books. The symptoms are very vague and indefinite, and there are none which are pathognomonic. So, frequently the disease is not recognised in its early inception, not in fact till it is well developed, and often not even until the surgeon's knife is called into play. In the present day exploratory operations are so simple, and afford such excellent opportunities for the surgeon interfering with the work of the physician, that I am afraid we

do not always cultivate our various senses to the extent that we should do, otherwise we would not so frequently require the assistance of the surgeon to complete our diagnosis. We can talk very glibly about the pathology and surgical treatment of tuberculous peritonitis, but I am inclined to think that if we recognised the disease and properly treated it in an early stage there would be fewer cases requiring operation.

The frequent failure to appreciate the development of such an important affection is, I think, partly due to the prevalent idea that peritonitis is a disease attended by severe and well-marked symptoms; whereas we know that in tuberculous peritonitis the onset is very insidious, and often there is little or no inflammatory reaction, so that in many cases tuberculosis of the peritoneum would be a more appropriate designation. No doubt in many cases there is a considerable amount of inflammation, but rarely if ever to such a degree as that associated with the introduction of other micro-organisms, such as the bacillus coli, streptococci, and pneumococci. There is none of that collapse associated with paralysis of the splanchnic area, such as occurs in perforation of the bowel with escape of its contents into the peritoneal cavity, and which makes such a permanent impression on the sensorium of the observer that he is apt to overlook the milder and more chronic form of tuberculous peritonitis.

This affection may occur at almost any age, but is rare under three and over fifty, and as a rule its existence is the more readily recognised or at least suspected the younger the patient. Its incidence is about equal in both sexes. The disease may be acute or chronic, primary or secondary. The acute and primary are rare affections, while the chronic and secondary are comparatively common. The acute form is usually part of a general acute miliary tuberculosis, and as there is no true inflammatory reaction of the peritoneum the symptoms are submerged by more pronounced signs of disease in the lungs and bowels. However, if the patient live long

enough attention may be drawn to the peritoneum by a slight increase in the size of the abdomen with general emaciation. There may be a small amount of free fluid in the peritoneal cavity, as in these cases the adhesions consist of a slight amount of lymph gluing the coils of bowels together. The abdominal walls become rather tense, though there can scarcely be said to be any severe pain or tenderness, and the respirations are thoracic. The temperature is usually rather high and may be hectic, but that depends on the involvement of other organs rather than of the peritoneum. If meningitis terminate the scene the scaphoid abdomen does not occur. The primary form is extremely rare, though like meningitis it may occur. As a rule, an original focus can be discovered at the autopsy, most frequently in the bowel, or generative organs and their adnexa. I have myself seen the origin of a general acute miliary tuberculosis in a caseous testicle, and those who treat female diseases know what a frequent source of general infection may be found in the tubes and ovaries. The symptoms in these primary cases in no way differ from those in chronic cases, which I am about to describe.

In chronic tuberculous peritonitis the symptoms usually begin very insidiously, and as commonly the lungs, pleuræ, or bowels are affected our attention may be drawn away from the peritoneum, though in fact these primary lesions should drive our observations more closely to the significance of an enlarging abdomen.

Just as in tuberculosis of the lungs when tuberculous ulceration of the bowel follows, the prominent chest symptoms subside; so when tuberculous ulceration of the bowels is followed by peritonitis, the diarrhœa and attenuated abdomen may be replaced by constipation and slight abdominal intumescence. This latter condition may give a rotundity to the abdomen, which taken in conjunction with the cessation of diarrhœa may give rise to the erroneous impression that the child is improving, whereas in reality the disease is extending. Pain is by no means

a prominent symptom in any stage of the disease, but usually a certain amount of tenderness can be elicited on manipulation. There may not be sufficient to give rise to any expression of pain beyond that depicted on the countenance of the dull and listless patient. He is careful in his movements to avoid all jars and knocks, and when in bed is apt to lie huddled-up, provided the abdomen be not much distended. These facts, coupled with thoracic breathing, which is an early symptom in all such cases, should arrest the attention of every intelligent observer at an early stage in this affection. At first the appetite may be good, but after a little it becomes capricious, and there may be nausea with occasional vomiting. I have seen the nutrition good till a fairly advanced period, but, as a rule, while the abdomen enlarges the rest of the body rapidly emaciates, the subcutaneous fat disappears, and the skin of the tense abdomen becomes thin and shiny so that the blue veins become very evident. Anæmia is usually marked, though this may be associated with a pink flush on the cheeks. The salivary secretion diminishes, the mouth becomes sticky, the tongue red, glazed and denuded of epithelium, and at this stage there is considerable thirst and no appetite. Constipation and improperly digested food give rise to griping and often moderately severe colicky pains, which are relieved by diarrhœa, and this may again alternate with constipation. As the disease advances the abdomen enlarges and this is specially marked in the upper half, the umbilicus protrudes, the recti and other abdominal muscles become very tense, and the breathing is purely thoracic. The general weakness, pain and tenderness increase, and the patient is confined to bed. The temperature is never very high, rarely rising much above 101° F. in the evening, and may often be subnormal, but the morning fall, even in cases of low temperature, is usually more characteristic than the evening rise.

At this stage a certain amount of free or loculated fluid may be detected. The large and tense abdomen exhibits various degrees of resistance at different places. Coils of

bowel being usually glued together, prominent tense swellings of knuckles of bowel containing gas and fæces can be readily felt. Commonly, the omentum is very much thickened, and owing to the peristaltic movements of the distended bowel it gets curled up on itself and forms a large, thick, sausage-like mass stretching across the abdomen about the level of the umbilicus. The amount of effused fluid is usually small, and its location depends on the extent of the peritoneal adhesions. In some cases it is fairly free, the flanks are dull, and a succussion wave is easily felt. In other cases there are numerous loculi forming evident cysts, which in cases of females have not unfrequently been mistaken for ovarian cysts. I have seen a large collection of purulent fluid walled off in the lower half of the abdomen, and as the bowels are floated up on the surface of the fluid the collection most frequently takes place below the level of the umbilicus. Œdema of the lower extremities and dependent parts of the abdomen occasionally takes place, especially in adults, but this is due to the feeble and atrophic heart rather than to any excessive abdominal tension. The pulse is frequent, small and feeble. The free edge of the liver can be felt about an inch or more below the costal margin. The spleen is usually rather enlarged but is not felt below the costal margin.

As the disease advances the emaciation becomes intense, the eyes are sunken, the temples and cheeks become hollow, the countenance shows evident signs of distress, the ribs stand out prominently, the lower ribs are partly everted by the large and prominent abdomen, and the breathing is thoracic. The skin over the chest may be dry, harsh and shrunken, while that over the abdomen is thin, smooth and shiny. The superficial veins are very apparent; the umbilicus is unfolded and the recti muscles separated. The patient lies on his back or side with his knees drawn up, weakness is great, and every movement causes pain. There is often stomatitis with a glazed red tongue and small ulcers on it and on the inside of the buccal cavity.

The bowels are often loose, the motions being of a dark, watery, offensive character, and where there is much intestinal ulceration there may be blood and mucus. There is often severe griping and colicky pains accompanying the diarrhœa, but, on the other hand, cases of fatal obstruction have been observed. The temperature in late stages frequently reaches 103° F. or 104° F. in the evening.

Improvement in these advanced cases rarely takes place, but I have seen recovery after the evacuation of an abscess which pointed at the umbilicus, and in another case after a fæcal fistula had formed in the same situation. One of the most ominous symptoms is the occurrence of purpuric spots on the extremities and flanks.

Diagnosis.—The diagnosis largely depends on a *résumé* of the symptoms which I have detailed. The slow and insidious onset, great wasting, abdominal distension, pain and tenderness, variable tension in different parts of the abdominal walls, a thin stretched skin with prominent veins over the abdomen and lower ribs, and in advanced cases a protruded umbilicus and separation of the recti muscles. Irregular lumps in the abdomen, especially a hard, firm, sausage-like mass at the level of the umbilicus from a thickened omentum are common conditions.

There may be general fluctuation or evidence of cyst-like collections of fluid. The breathing is thoracic. The temperature is variable and generally marked by a morning fall. The patient looks ill, is dull and listless but always intelligent, and his senses are normal. The presence of tuberculous disease in other organs assists the diagnosis. The large tympanitic abdomen which occurs in rickets and dyspeptic troubles is easily distinguished by the uniform and variable distension, absence of muscular rigidity, and no great variations in the resistance at different parts, no evidence of fluid, and thoracic breathing not a prominent feature.

In typhoid fever the abdomen is often distended, but the tension is not great and there is no muscular rigidity. The spleen is large and easily felt, the patient is dull and

apathetic, his intelligence and all his senses are blunted. There is continuous fever, and other typhoid symptoms which need not be enumerated. In cases of ascites arising from cirrhosis and Banti's disease the accumulation is great, the fluid is clear and of low specific gravity. In these cases there is often localised peritonitis with well-marked friction fremitus, and in some cases the differentiation may cause some difficulty. The same may be said of simple chronic adhesive peritonitis arising from other causes. In some such cases I have found the injection of the peritoneal fluid into guinea-pigs of some value. As a rule, patience and careful observation soon settle the diagnosis.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the 12th*, 1902.)

16. TUBERCULOUS PERITONITIS: SYMPTOMS AND DIAGNOSIS.

By GEORGE CARPENTER, M.D.

Symptoms.—Tuberculous peritonitis is met with in practice under several fairly well-defined clinical conditions, and it is therefore possible to arrange these cases with some method. These clinical conditions are :

1. A matted condition of the bowel, which is often, but by no means necessarily associated with tuberculous intestinal ulceration and caseous disease of the mesenteric glands ; a disease called tabes mesenterica, which has the well-known clinical features of a tumid abdomen, abdominal pain, diarrhœa, wasting, and sometimes fæcal fistula at the umbilicus.

2. A condition in which a child is out-of-health, has a full stomach, and complains of griping pains there. Under examination, hard and fixed lumps are felt in one part or

another of the abdomen, usually close under the parietes, but not always so. The disease is due to the luxuriant growth of miliary tubercles in large masses. This will often occur in the omentum, which becomes much thickened and contracted into a thick band, and stretches horizontally across the upper part of the abdomen. Sometimes a large mass, or plate, forms between the liver and the diaphragm, but here of course not recognisable clinically. At other times the omentum forms a thick apron, which lies in front of the intestines, and is adherent to them, and to the liver, spleen, and abdominal parietes. Such a thickened mass often has areas of varying density and induration, which may be felt as isolated nodules of superficial-lying thickening underneath the abdominal wall. Or, a mass may form in Douglas's pouch which can be detected on making a rectal examination. Again, tuberculous masses may be felt in any part of the abdominal cavity, and these are mostly intestinal coils matted together with tuberculous deposit outside the bowel, a fact which explains a clinical feature that is often puzzling, viz., that the tumours are constantly varying in size and definition. There is also another practical point of some interest and that is, the bowel may be obstructed by the tuberculous deposit enveloping it, inducing tormina, borborygmi, and obvious peristaltic movements of the greatly distended gut which terminate at the seat of obstruction and perhaps occasion thereby a group of symptoms which may possibly lead to the suspicion of chronic intussusception. *The tuberculous tumour*, the most common variety of tuberculous peritonitis, is not often associated with much ascites. In the course of the complaint a certain amount of fluid may come and go, but the quantity is not often great, though it may be so large as to mask the underlying mischief.

Fixed lumps may be due to caseation of the mesenteric glands alone. Such glandular masses may be so matted together as to form a large fixed tumour, or they may retain their original isolated positions and be felt through

the abdominal wall, and bimanually *per rectum*, as a characteristic grape-like mass. Such cases often find their way into the wards of a hospital, not for this complaint but as instances of tuberculous meningitis, or of broncho-pneumonia, or of a child wasted to a skeleton and in a state of collapse from diarrhœa. Sometimes their abdominal condition is recognised during life, but intestinal ulceration and caseous mesenteric glands often escape detection, and are found in the post-mortem room. In a girl 10 years old, a fixed tumour was due to the following condition. The morbid changes were limited to the small intestine, commencing about two feet above the ileo-cœcal valve. Here, and for some little distance down to within six inches of the valve, there was extensive intestinal ulceration together with caseation and matting of neighbouring glands, the whole being knotted together by dense adhesions. Some of the ulcers were of cartilaginous hardness. The rest of the abdominal cavity was quite free from disease. There was secondary tuberculous lung disease. The limited extent of the disease suggests the idea that it might have started in a mild attack of enteric fever. A microscopical specimen in the possession of Dr. Alfred Lingard, Imperial Bacteriologist to the Indian Government, is in favour of such a theory. It was taken from one of the mesenteric glands of a case of typhoid fever, and shows typhoid and tubercle bacilli side by side. In some instances, when an opportunity has presented itself of making a post-mortem examination of cases occurring in this group, I have also found caseation of the mesenteric glands and intestinal ulceration, and such are clinically allied to the group of cases occurring under heading No. 1.

The amount of febrile disturbance is not great, although exceptions do arise and well-marked instances of a remittent type of fever are seen. A fairly constant feature is the large proportion of subnormal temperatures encountered in this disease. Day after day the temperature may be found ranging between 98° F. or 97° F., or even lower, with occasional rises to 99° F. or thereabout. These cases

often linger on week after week, and month after month without any marked alteration either way, and frequently leave the hospital improved in regard to their general health, though the tuberculous tumours remain much as before.

Under heading 3 are cases that come under observation as examples of *ascites*. Throughout their illness the condition may begin and end as one of ascites. Perhaps, a rectal examination may reveal some slight thickening in the abdominal cavity which would have escaped detection by the ordinary methods of examination. Ascites may mask the condition of tuberculous tumour just mentioned, and it is not until a paracentesis abdominis has been performed, or the fluid has become absorbed, that the underlying mischief is detected. The converse may also happen, but this is not so common. As an uncommon sub-variety may be mentioned that condition in which fluid is encysted amongst the intestinal coils, and under this variety may be grouped cases which come under notice as *localised tuberculous abscesses*. Variations are sometimes noticed in the character and behaviour of the abdominal contents. Thus, in a case of abdominal distension, sometimes there may seem to be a large quantity of fluid, and at other times the contents appear to be chiefly gaseous. Such variations succeed one another quickly. When fluid seems to be present in the abdomen and behaves in the manner described, it is probable that fluid is really there, and that instead of being definitely encysted in one particular locality or free in the peritoneal cavity it is held by the intestinal coils, and that as these alter in position and distension so the evidences of the fluid alter and alternately appear and disappear.

The febrile symptoms and fever encountered in ascites are not marked. The temperature may be raised for a day or two to 100° F., or to 101° F., and usually quickly subsides. On the other hand, a hectic type of temperature may be found in those who ultimately recover.

4. There is a type of tuberculous peritonitis in which

the abdomen is greatly distended, tense, and drum-like, and offers great resistance to manipulation. It may happen that nothing abnormal can be felt by a bimanual examination, but, on the other hand, there may be a suspicion of peritoneal thickening, and sometimes dull areas can be mapped out on percussion of the abdominal walls, quite apart from obvious thickening. Given, fever of an intermittent type, pea-soup-like motions, possibly some splenic enlargement, moist sounds in the chest, drowsiness and some delirium, all of which may happen in combination in such a case, and it is not difficult to mistake the condition for that of enteric fever. Subsequently there may be effusion of fluid, or definite lumps may make their appearance, or the bowels may be found to move under the hand *en masse*. On the other hand, the primary condition may clear up without any further developments. In infants and young children, the condition which is most likely to be mistaken for it is that of chronic gastro-intestinal catarrh from improper feeding; a bimanual examination may in such a case prove decidedly helpful.

The foregoing remarks are the outcome of a study of 91 cases of tuberculous peritonitis which have been under the writer's care during past years. Of these cases 55 were males and 36 were females.

Phthisical history.—In 32 of these cases there was a family history of phthisis or other tuberculous affection; in 29 cases there was no history of such; and in 30 cases the notes were not sufficiently precise on the subject to make them of value. About half the number of cases, where an inquiry had been made, gave a history of tuberculous affections.

Age incident.—Their ages are as follows:

Months.	Cases.	Years.	Cases.
3 to 6	2	1½ to 2	4
6 „ 9	0	2 „ 3	15
9 „ 12	2	3 „ 4	19

Months.	Cases.	Years.	Cases.
12 to 16	8	4 to 5	4
		5 „ 6	12
		6 „ 7	3
		7 „ 8	5
		8 „ 9	5
		9 „ 10	3
		10 „ 11	5
		11 „ 12	3
		12 „ 13	0
		13	1

Total 91 cases.

A little less than $\frac{1}{6}$th of the total number of cases arose within the first two years of life. From this period to within 6 years of age there were no less than 50 cases, or close on $\frac{5}{9}$ths of the total number of cases; the disease appears, therefore, to be more common about this period of childhood than at other times.

Enlargement of the liver.—During and within the first two years of life 4 cases, or $\frac{1}{4}$th of the number, were associated with an enlarged liver; one child died and its liver was found to be "nutmeg." From 2 years to within 6 years of age there were 5 cases, or $\frac{1}{10}$th of the number; one died and its liver was found to be fatty. Of these cases, in every instance but one, the spleen was also enlarged.

Enlargement of the spleen.—During and within the first two years of life there were 5 cases with enlarged spleens, or $\frac{5}{16}$ths of the cases; one of them died. From 2 years to within 6 years of age there were 8 cases of splenic enlargement, or $\frac{8}{50}$ths of the cases, and 3 of these died. Of the deaths one was that of a child just over 2 years old; its spleen though enlarged was free from tubercles. In the other instances the enlarged spleen contained miliary tubercles. The most prolific periods for splenic enlargements from all causes are firstly during the first eighteen months of life, and secondly, and to a less extent,

from then up to the fourth year.* At these ages splenic enlargements are quite common. Splenic enlargements associated with tuberculous peritonitis have been, in my experience, infrequent and uncommon, in comparison.

Phthisis.—This occurred in 16 cases, and in 4 of them within the first two years of life. Of the 4 cases, 3 died and in one there was caseous pneumonia, and in another miliary tuberculosis. Of the 12 cases, 9 died and in 4 of them there was miliary tuberculosis of the lungs. In my experience, obvious phthisis has not been a common clinical complication.

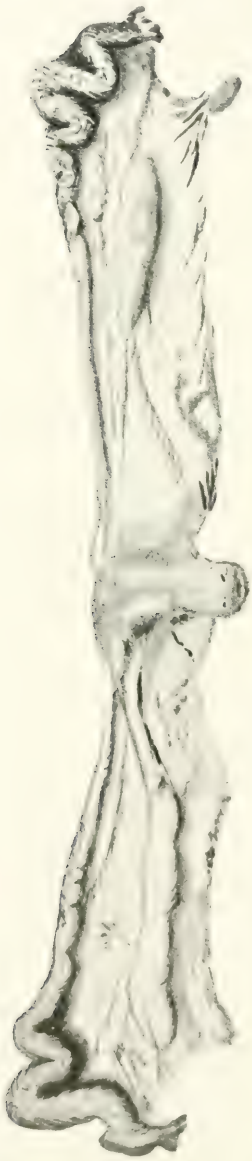
Pleurisy.—Of this complication I possess but one clinical record, viz., that of the child 3 years old, previously mentioned, who succumbed to empyema.

Tuberculous disease of the male genitalia.—Five of the cases of tuberculous peritonitis were associated with the above complications. In a boy of 11 years old the vesiculæ seminales were tuberculous. In a boy, aged 5 years, there was tuberculosis of the seminal vesicles, the prostate, and the body and epididymis of one testicle. A boy of 6 years old had caseous epididymitis with a corresponding caseous seminal vesicle. In a boy of $2\frac{1}{2}$ years the seminal vesicles and the prostate were tuberculous. In a child of 18 months old both seminal vesicles were tuberculous, and the epididymis on one side.

Tuberculous disease of the female genitalia.—My records of this complication have been gained by the use of combined rectal and bimanual examinations during life, and also in the post-mortem room. In a child, aged 9 years, the Fallopian tubes were enlarged and caseous. Tuberculous extension had taken place from the peritoneum, and they had been invaded at their fimbriated extremities (Fig. 10). In a girl, aged 4 years, the ovaries were matted to the Fallopian tubes, the extremities of which showed cavities filled with caseating material; she had

* *Vide* 'Syphilis of Children,' by George Carpenter, M.D., pp. 48 and 49, diagram 19; also "Splenomegaly in Infants and Young Children," 'Reports of The Society for the Study of Disease in Children,' vol. iii, pp. 296—317.

Fig. 10.



tuberculous intestinal ulceration, together with tuberculous brain tumours, but no peritonitis. In a girl of 7 years I found an enlarged uterus, the right Fallopian tube was the diameter of a lead pencil, and there was a tuberculous lump the size of a pigeon's egg just above the top of the sacrum. I subsequently lost sight of her. A girl of 7 years old, who had tuberculous ascites, had a rounded tumour, the thickness of the index finger and three inches in length, attached to the top of her uterus. This tumour subsequently disappeared. She is a healthy woman now. In a child, aged 2 years and 2 months, there was considerable peritoneal thickening in the hypogastric region and its neighbourhood, and the left ovary, which I found to be enlarged, was attached to this. This child had tuberculous glands in the neck and an enlarged spleen also. In a child, aged $2\frac{1}{4}$ years, there was a tuberculous lump involving the right lumbar, iliac, and the hypogastric regions. The left ovary and Fallopian tube were healthy, but the mass had invaded the tube and ovary on the corresponding side. In a child, aged 14 months, there was a hard

lump occupying the umbilical, and hypogastric regions, and the right ovary was connected with it. In a girl, aged 6 years, there was a tuberculous lump in the right inguinal, lumbar, hypogastric, and umbilical regions, together with

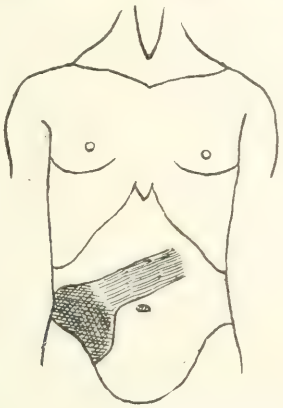


FIG. 11.—Condition of the abdomen as determined by palpation.

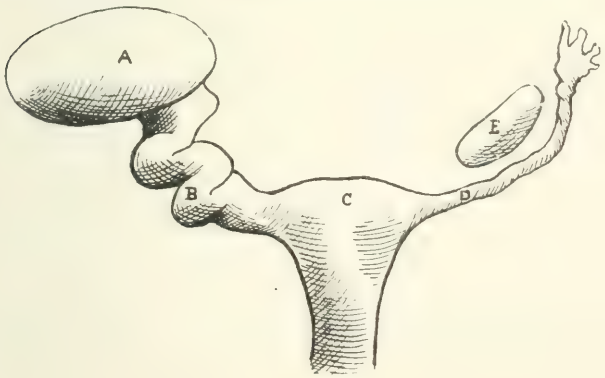


FIG. 12.—Diagrammatic condition of the internal female genitalia as determined by a rectal and bimanual examination.

a typical omental thickening. The uterus was normal. Passing from it to the right was a curly tube, the size of the little finger, which could be hooked down, when an oval lump was felt, which was undoubtedly an enlarged ovary (Figs. 11 and 12). The left Fallopian tube was not so en-

larged, and it did not curl; its corresponding ovary was not enlarged. In a girl, aged 3 years, the abdomen was lumpish, and this lumpishness extended on the right side to the inguinal region. The uterus was normal. The right Fallopian tube was the size of a penholder, and it could be traced into a thickened and hardened mass, which was possibly the tube and ovary combined, and the whole was finally lost in the abdominal lumpishness previously mentioned.

In a girl of 14 months old there was lumpishness

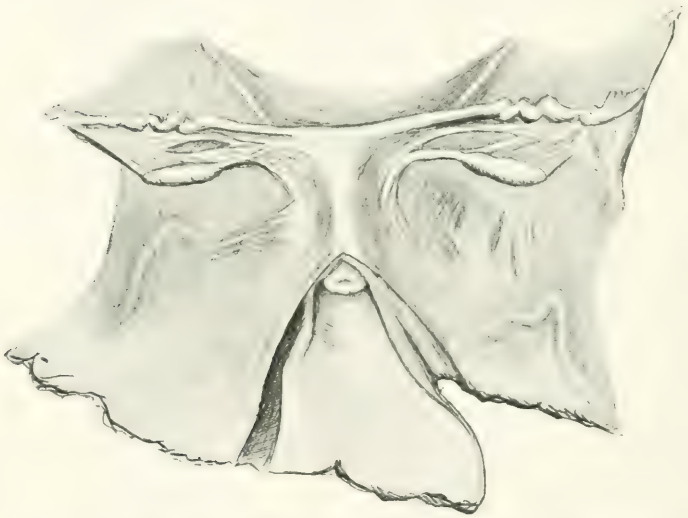


FIG. 13.—Normal pelvic organs of a child aged 2½rd years, reduced one-fourth. The vagina has been laid open from behind, and shows the os uteri. On either side of this is the edge of the falciform ligament with its uterine attachment, and to right and left of the falciform ligament the ureters are seen as two curly tubes. The round ligaments are depicted in front of the Fallopian tubes, and are ill-developed.

in the epigastric, umbilical, hypogastric, left iliac, and lumbar regions. The uterus and right ovary were enlarged. The left ovary and tube merged into the abdominal lumpishness.

In a girl, aged 1 year, there was lumpishness in the umbilical, lumbar, iliac, and hypogastric regions. The right Fallopian tube was rounded and the size of a cedar

pencil; it ended in the abdominal lumpishness. The corresponding ovary could not be detected. The left Fallopian tube was much larger, but not quite so distinct as that on the other side. It ended in a smooth lump, the size of a walnut kernel, which was probably the ovary. The uterus was not enlarged.

I have had no experience, clinical or pathological, of primary tuberculous infection of the uterus and the adnexa in children.

Tubercle of the choroid.—My examinations of the eyes in these cases have not been systematic but I have made a considerable number of negative observations and in three cases I found tubercles in the choroid. One, a female child, aged 4 years, with caseous Fallopian tubes and tuberculous brain tumours, previously mentioned, had bright tubercles at the yellow-spot regions. Another, a male aged 2½ years, with intestinal ulceration and caseous mesenteric glands and miliary tuberculosis, had double optic papillitis and a large tubercle at the yellow-spot region.* A third, aged 4½ years, with intestinal ulceration, caseous mesenteric glands, tuberculous peritonitis, and tuberculous meningitis, had double optic papillitis and a choroidal tubercle in the right fundus oculi.

Tuberculous meningitis.—This has not been a common complication, in my experience, but two of the fatal cases developed it. In one of the cases in which it occurred the abdomen became retracted.

Unclassified.—Occasionally acute peritonitis supervenes on the chronic; this happened in one case of omental tuberculosis. Protrusion of the umbilicus is seen in some cases, as also a red blush or pink halo round it. Fæcal fistula is not a common occurrence. Enlarged abdominal veins are the rule, and occasionally there is some slight œdema of the abdominal walls.

Diagnosis.—Next arises the question of diagnosis and,

* *Vide* 'Reports of The Society for the Study of Disease in Children, vol. i, pl. viii, fig. 4, article, "Tuberculosis of the Choroid," by George Carpenter, M.D., and Sydney Stephenson, C.M.

before all things, I would strongly urge the importance of rectal exploration associated with bimanual examination, either with or without an anæsthetic. A full account of the method will be found in 'Pediatrics,'* in 'The Scalpel,'† and in 'The British Gynæcological Journal.'‡ By means of an examination conducted in this way not only can the whole of the true and false pelvis be explored with its contained organs, but in young children, from birth to 4 or 5 years of age, it is possible to examine a considerable area of the abdominal cavity outside the pelvis. Not only can abdominal lumps and tumours generally be explored with greater ease by such an examination, but it is also possible to detect an amount of peritoneal thickening which would escape the most careful abdominal examination conducted in the ordinary way. In the healthy abdomen, the finger in the rectum and those on the abdominal wall should be separated by what appears to be the thickness of the abdominal wall merely, but when there is peritoneal exudation the separation of the examining fingers is often found to be more than that; thus, in the same case, one part of the abdomen may be found normal and another appreciably thickened. Comparison here comes in useful. Before such an examination is undertaken an enema should be given, the bowel cleared of its contents, and the bladder emptied; this will facilitate examination and prevent errors in diagnosis. Difficulties not infrequently occur in differentiating tuberculous conditions from other abdominal diseases. Thus, a boy, aged 14 years, was suddenly seized with pain in the abdomen, seventeen days previously, thought to be due to eating a holly-berry. For seven days and nights he suffered from pain and vomiting, and on the third day he passed a "pint of dark blood" by the bowel, and blood

* "On the Value of Rectal Exploration as an Aid to Diagnosis in Diseases in Children," by George Carpenter, M.D., June 1st, 1896, pp. 481 to 507.

† July and August, 1896.

‡ May, 1896.

appeared in his stools for two succeeding days. He had a daily evacuation of the bowels, until within two days of his attendance at the hospital. He was losing flesh very rapidly. When seen he was pale, emaciated and evidently ill. The abdomen was easily manipulated. About two fingers' breadth above the umbilicus a resistent, sausage-like tumour, a trifle tender on pressure, extended transversely across the abdomen. A simple soap enema was administered and this brought away formed fæcal masses, but no blood. A rectal examination revealed nothing abnormal. His organs were healthy. For a fortnight he passed natural motions, and was for the most part free from pain. The tumour was then not so hard, or so distinctly rounded. He was emaciating. For another period of fourteen days he was occasionally sick, and abdominal pains were a variable feature. His bowels acted regularly after enemata. In two or three days from then acute symptoms supervened, and it was thought necessary to perform laparotomy. An ileo-colic intussusception was found, which was reduced, but he died within a few hours. Intussusception was suspected at the time he first came under notice, but this was negatived on the following grounds, 1, the emaciation; 2, the flaccidness of the abdomen; 3, the passage of formed fæces; 4, the situation of the tumour, and the absence in it of contractions, which were carefully sought. The suspicious feature of the case was the passage of such a quantity of blood; blood in the stools is not common with tuberculous intestinal ulceration.

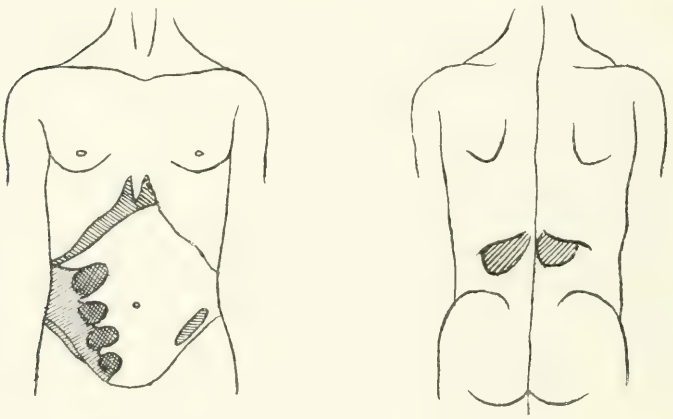
A girl, aged 3½ years, who was suffering from whooping-cough and broncho-pneumonia, made satisfactory progress in regard to her illness during a period of three weeks. Then she complained of abdominal pain; she was slightly sick at times, and retched continually. Her legs were drawn up, and her face was pale. She had not passed a motion for two days, but on the day following the attack she had a slight action of the bowels attended by abdominal pain. An abdominal examination revealed a

collection of several rounded masses in the abdomen, manipulation of which caused a little pain. The abdomen was perfectly flaccid. The mass was chiefly centred round the umbilical region, its lower limit being the last lumbar vertebra and its upper was bounded by the top of the umbilical region. The tumour was semi-elastic and gave the impression of enlarged mesenteric glands. She suffered greatly from abdominal pain and frequently vomited, but passed neither blood nor mucus by the bowel. The tumour was free from intestinal contractions. She was collapsed and lay on her back with her legs drawn-up. After a rectal examination she passed a loose motion, associated with some formed material. Abdominal section was performed. She had an intussusception which was limited to the small intestine. She died half an hour after the operation. In this case the acuteness of the onset, the sickness, the great abdominal pain, and the collapse were unlike tuberculous peritonitis, though the physical signs were in favour of it. Take another illustration. An infant boy, aged 11 weeks, was sent to me with the following history. Since three weeks of age he had not passed anything solid from the rectum, only black fluid which was very offensive, and there had been frequent vomiting of what was stated to be fæculent matter. A fortnight before I saw him he had three fits. He was a pale-faced, poorly-nourished, weakly-looking infant, with a distended abdomen over which the cutaneous veins were very pronounced. Abdominal palpation did not afford any information, so I made a rectal exploration. Situated in the right inguinal, hypogastric, and umbilical regions was a freely-movable, smooth, oval tumour, from 3 to 4 inches long and from $1\frac{1}{2}$ to 2 inches broad, and roughly estimated about the size of a kidney, indeed there was some suspicion as to the presence of a hilum, though I could not be certain about that. The kidneys could be felt in their normal positions. A sense of fluctuation in the tumour could not be appreciated, with absolute certainty. Several enlarged glands, varying in size from

a pea to an almond, were felt about it, and one was behind it. Attached to it were some small nodules. To the right of the tumour the intestines felt contracted and empty, rather than infiltrated with inflammatory material. Was it a tuberculous abscess? The enlarged glands were certainly suspicious. An abdominal section was performed, and the tumour was found to be a mesenteric cyst firmly attached to the intestine. Two ounces of fluid were withdrawn from it, which contained white and red blood-corpuscles and $\frac{1}{2}\frac{1}{1}$th of albumen. Several supposed tuberculous glands were also found in the mesentery at the time of operation; but the child died and on post-mortem examination the glands were found not to be tuberculous. Here is another interesting case. A boy 2 years old, who was brought to the Evelina Hospital for whooping-cough, was found to have broncho-pneumonia and an abdominal tumour. A combined rectal and bimanual examination led me to form the opinion that the abdominal tumour was a horse-shoe kidney, and the child was admitted into the wards with that diagnosis. There, those in charge were equally positive it was a case of tuberculous peritonitis. The child subsequently died, and the original diagnosis was then proved to be correct. Abdominal malignant disease offers considerable difficulties to diagnosis. In malignant disease there is the same rolling-up of the omentum, when this part is attacked, and tumours, similar to the touch to those met with in abdominal tuberculosis, may be felt through the abdominal walls. A girl, $3\frac{1}{2}$ years old, had been ailing for three months, and three weeks before she was examined her abdomen began to swell. On examination it was enlarged, measured 21 inches at the umbilicus, and was dull in the flanks. This dulness did not shift on change of posture. Large blue veins coursed over its parietes, which were flaccid. Lumps were detected chiefly on the right side, and here there was a rounded and nodular mass (*vide* Fig. 14) which was situated in the right inguinal region. In the right nipple line, some little distance below the costal margin, was

another rounded mass. These rounded tumours were united by ill-defined thickening. In the left iliac fossa there was a tumour the size of a walnut, which was thought to be the ovary. Behind, $\frac{1}{2}$ an inch below the 12th rib on either side, could be felt smooth rounded masses, which were considered to be the lower ends of the kidneys. Her feet and legs were œdematous; the urine was normal. She was under treatment for 62 days, and finally died of exhaustion. At the necropsy a tumour was detected as described arising from the right iliac fossa. The intestines were turned aside. The lower mass

FIG. 14.



was found to involve the cæcum; it formed a distinct tumour there, and at the same time diffused into the adjoining mesentery. It bound down several coils of intestine, and one especially thick deposit passed towards the rectum. The upper swelling was a round, well-defined growth of the size of a small orange, situated in the mesentery adjoining the intestinal growth. The left ovary was replaced by new growth. A new growth, the size of a broad bean, was found in the wall of the right ventricle. The tumours were small-round-celled sarcomata. Retro-peritoneal sarcomata may also occasion difficulties and l

recently published in the 'Reports' a case of sarcoma* with implication of the mesenteric glands, which could not be distinguished clinically from tuberculous peritonitis, save that it did not react to tuberculin. Further vaginal polypi (sarcomata), of which there are some few cases on record, with secondary implication of the pelvic organs and their neighbourhood must be borne in mind.

There are cases of simple effusion into the peritoneum no doubt, allied to the similar conditions which occur in the pleural cavities, though I cannot call to mind, in a very large acquaintance with simple pleural effusions, an instance of ascites which was associated with pleural effusion. I have seen purulent peritonitis, associated with empyema, which was non-tuberculous and also simple purulent inflammation of the peritoneum apart from empyema of which the following case is an example.

A male infant, aged 11 months, attended the Evelina Hospital with the following history. He had suffered for 14 days from severe abdominal pains accompanied by nausea and vomiting, and he had lost flesh. There was a remote history of consumption on the maternal side. When seen he was pale, but fairly nourished. The abdomen was distended and prominent. Around the umbilicus was a well-marked local projection, which was reddened, tender to the touch, and considerably indurated. There was dulness in the umbilical, left inguinal, lumbar, and hypochondriac regions, but no fluid thrill could be obtained. The following week there was no material change, though if anything the abdomen was not quite so swollen.

About a fortnight later pus appeared at the navel, and he then developed a fæcal fistula, large loose yellow motions, mixed with pus, passing from it. He subsequently died. On opening the abdomen a large abscess cavity was seen, mostly situated to the left of the umbilicus, and stretching across the middle line to the right iliac fossa.

* "A Case of Supra-renal Sarcoma," by George Carpenter, M.D., vol. ii, pp. 69—73.

Beneath this lay many coils of intestine, which were not matted together. On slitting-up the intestines, the mucous

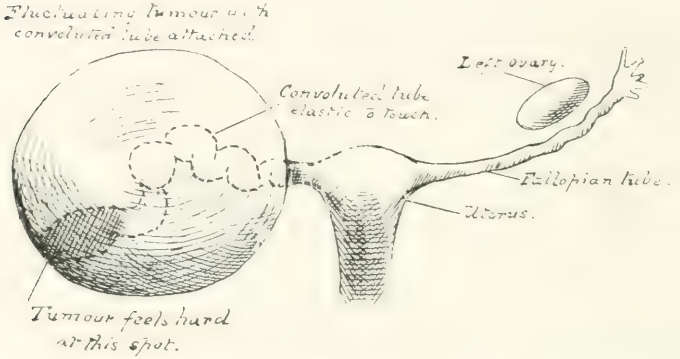


FIG 15.—Condition of the internal female genitalia in a child 22 months old, as determined by a rectal and bimanual examination.

membrane immediately surrounding a perforation which led into the abscess cavity from the upper part of the ileum

FIG. 16. Drawing of the ovarian tumour and dilated Fallopian tube from a child 22 months old, as it appeared after removal.

was found to be perfectly healthy. The abscess evidently opened into the bowel. The organs were healthy.

I have also notes of a case of non-tuberculous peri-

tonitis in a boy, aged 7 years, who after many months of illness, associated with abdominal abscesses which cropped-up in various situations, finally made a good recovery and has now reached a healthy adolescence.

There are other abdominal conditions which may occasionally give rise to a mistaken diagnosis. In young children the urachus and obliterated umbilical vein may be felt through the abdominal wall as indurated cords—a knowledge of this will prevent error. Ovarian tumours, cystic, malignant, and dermoids may also prove puzzling, and rectal exploration should here prove of value. I have diagnosed an ovarian cyst with tubal implication in a child 22 months old, and this was verified by a surgical operation, (*vide* Figs. 15 and 16).

The full abdomen seen in infants and young children suffering from gastro-intestinal catarrh may, on inspection, suggest the idea of tuberculous peritonitis, but a rectal exploration should here render valuable assistance and prevent any error in diagnosis. Appendicitis, which is comparatively infrequent in children, has but to be borne in mind to prevent mistakes; and abdominal hydatids, of which I have seen several examples in children, may occasion difficulties, but the use of the exploring needle should prevent any confusion in that direction.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the 12th,* 1902.)

17. TUBERCULOUS PERITONITIS: SYMPTOMS AND DIAGNOSIS.

By J. PORTER PARKINSON, M.D.

I wish to limit myself to-day to one or two practical remarks with regard to the symptoms and diagnosis of this

disease. On looking over the notes of some 22 cases from the wards of the North-Eastern Hospital for Children and the London Temperance Hospital which have occurred during the past year, I was struck with the somewhat rapid onset, and the absence of previous abdominal symptoms. The average history of 19 cases only extended over 3½ weeks, while in two cases the patient had been ill for 5 months and in the other for a year. This is interesting, if compared with the usual lengthy history of the majority of cases of tuberculosis of the lungs.

Again, all the cases appeared to be suffering from the symptoms of peritonitis for the first time, except in one case where more or less trouble had existed for a year previously.

A sign which I have not seen previously noted was the occasional presence of enlarged veins over the upper zone of the abdomen, above the level of the umbilicus, suggesting some interference with the portal circulation. This local venous enlargement was certainly present in three of the cases as shown by the notes, and probably existed in others but has not been recorded. In all the cases in which this condition was noted there were to be felt nodular masses in the abdomen, and in two of them there was evidence of much shrinking and matting of the omentum and mesentery, which owing to its obstructive effect on the radicles of the portal vein, chiefly perhaps the tributaries of the superior mesenteric vein, caused dilatation of the same vessels as is produced by obstruction of the portal vessels in the liver. A case of tuberculous peritonitis, read by Mr. Raymond Johnson before the Clinical Society in 1898, in which there was thrombosis of the superior mesenteric vein supports this view.

Another point I should like to mention is the detection of small amounts of fluid in the peritoneal cavity. If this fluid is free and not loculated by adhesions, it may be easily recognised by placing the patient on his knees and elbows, so that the front of the abdomen is the most

dependent part and will give a dull note on percussion. This simple method is mentioned by Osler, but seems to be very rarely practised, as no house-physician I have yet met has seen it done. But, it is evident that much smaller amounts of free fluid can be detected by its means than by any other.

Finally, I should like to mention a disease which though rare may exactly resemble tuberculous peritonitis in its symptoms, and may make a positive diagnosis impossible, at least before laparotomy has been performed. I allude to colloid cancer of the peritoneum, of which I have at present an example under my care at the London Temperance Hospital. The patient, a girl aged 12 years, had been noticed to be ill for two weeks, suffering from abdominal distension and diarrhœa.

There was no vomiting and the tongue was clean.

On admission the abdomen was exceedingly distended with fluid, of which 8 pints were removed by tapping; this was straw-coloured, of specific gravity 1015. On boiling it completely solidified, and under the microscope there was a deposit of epithelioid-looking cells which showed numerous clear vacuoles in the protoplasm. After tapping, irregular masses were felt in the abdomen chiefly to the right and left of the umbilicus, and the spleen could be felt.

The child was anæmic and thin but not emaciated. The chest was normal, except that the heart's apex was in the 3rd intercostal space, pressed-up by the effusion.

The temperature was normal. The urine was concentrated and contained a slight trace of albumen.

As the fluid quickly reaccumulated Sir W. Collins kindly opened the abdomen to remove the fluid and if possible effect a cure, as the diagnosis was made of tuberculous peritonitis.

At the operation it was noticed that there were numerous nodular, greyish-white masses scattered all over the peritoneum, but they were translucent and somewhat unlike masses of tubercle.

One mass was removed for microscopical examination and showed typical colloid cancer.

With regard to the ages of the 22 patients I have alluded to, 14 were under the age of 4 years while the other 8 were aged from 4 to 12 years. Seven cases, or $\frac{1}{3}$rd of the whole, were between 3 and 4 years of age.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the 12th,* 1902.)

18. TUBERCULOUS PERITONITIS: PATHOLOGY AND MORBID ANATOMY.

By THEODORE FISHER, M.D.

The main features of tuberculous peritonitis are too well-known to need recapitulation, but having, through the kindness of Dr. Lauriston Shaw and Dr. Lazarus Barlow, been able to examine some of the post-mortem records of the hospitals with which they are connected, and having looked through also the records which are under my own care, I have been furnished with a few facts, which although they do not reveal anything new may be worthy of bringing briefly before your notice.

Before, however, referring to the leading features of the morbid anatomy of tuberculous peritonitis a brief comment may, I trust, be not out of place upon the etiology. At one time my experience led me to believe, with some of the American observers, that primary abdominal tuberculosis is extremely rare. Up till about three years ago, I had only met with one case of tuberculosis in a child where the bronchial glands were entirely free from disease, and it may be remarked, in passing, that unless the bronchial glands lying hidden within the lungs are examined a caseous focus in that situation may be overlooked. There is not the least doubt in my mind that abdominal tuberculosis can owe its existence to a

small tuberculous focus within the thorax. For example, I have seen acute tuberculosis limited to the peritoneum complicating a fatal case of rheumatic pericarditis, where the only caseous focus in the body was in a bronchial gland. The pericarditis, which was without doubt rheumatic in nature, had no doubt disturbed the circulation through the bronchial and mediastinal glands and caused dissemination of tubercle bacilli, which in this case attacked for some reason the peritoneum only. Again, in diphtheria I have seen numerous recent tubercles in the spleen and liver, but in no other organ of the body, the source of infection being there also a single caseous focus in a bronchial gland. Such cases as these show that abdominal tuberculosis may arise from a tuberculous focus outside the abdomen. While, however, I believe this to be true, I also now cannot doubt that primary abdominal tuberculosis occasionally, perhaps not very infrequently occurs, since I have met with at least three cases in which no disease in the lungs or bronchial glands could be found.

Most of the following remarks are based upon the examination of records of 80 cases of tuberculous peritonitis, 30 being from Guy's Hospital, 20 from the Westminster Hospital, and 30 from the Bristol Royal Infirmary. It may be added that all cases were excluded from the list in which the peritonitis appeared to have been merely a complication of marked tuberculous disease elsewhere, such as of the lungs, bones or joints.

Tuberculous peritonitis is commonly classified under the types of the ascitic, the fibrous, and the ulcerous. The fibrous form is sometimes divided into the dry and the adhesive varieties, but the adhesive is so much the more common that the name adhesive may almost be considered synonymous with the word fibrous. In reality also the so-called ulcerous is only a variety of the fibrous, and perhaps another variety of fibrous form might be added and called the cystic.

Of these different forms of tuberculous peritonitis the

variety most commonly met with is the fibrous. Fluid may have been present, perhaps in considerable quantity, at some time during life, but at the time of death general adhesions only are met with. Thus, of the 80 cases examined 48 showed general adhesions, whereas, in 8 only was the quantity of fluid present sufficient in amount to warrant the name ascitic being applied to the condition. In passing, it may be mentioned that while in the great majority of cases there are wide-spread adhesions which are fibrous and evidently of long standing, more recent adhesions of only partially organised exudations may be present, and occasionally the adhesions throughout the abdomen are of this character. The adhesions, however, whether fibrous or of less organised character are always intermingled with evidences of their tuberculous origin. These may be nothing more than scattered fibroid or caseous tubercles that generally stud the folds of peritoneum more thickly than the surface of the intestines, but as commonly there are caseous plaques, or caseous masses of various shapes and sizes, chiefly hidden away amongst the adhesions. It is suppuration in these caseous masses which has given the name to the so-called ulcerous form, since the masses when suppurating commonly open at more than one point into the intestines. Perforations of this character were present in eight cases, that is in 10 per cent. of the number examined. With these cases we may classify those in which perforation occurred from within the intestine, that is through the base of a tuberculous ulcer. There were 5 cases in which perforation had thus occurred.

While caseous masses are common in tuberculous peritonitis, it is somewhat interesting to note that very definite thickening of the great omentum proved to be comparatively rare. It is mentioned as being markedly thickened only seven times, and as having formed a tumour only twice. In my own notes more than once I have commented upon the absence of marked thickening in the great omentum, and other pathologists who wrote the

records which I examined have done the same. While, however, the great omentum need not be thickened, the presence of thickening may possibly sometimes be overlooked. The great omentum although thickened may be firmly adherent to the abdominal wall and be cut through and stripped off the intestine in opening the abdomen.

It has been mentioned that we might speak of a cystic or a fibro-cystic form of tuberculous peritonitis. In these 80 records of cases, however, this variety does not appear to have been very common. In five instances only were well-marked localised collections of fluid present. In two the fluid was purulent, in two serous, and in one fæculent. In two instances the collection of fluid had found an exit at the umbilicus.

Perforation of the intestine by tuberculous ulcers has already been referred to, but it may also be of value to note the frequency with which ulcers were present in these 80 cases of tuberculous peritonitis. It is definitely stated that they were absent in 30 cases, and that they were present in almost the same number, that is in 29. In the remaining 21 their presence or absence is not recorded, but when one considers how firmly adherent the intestines are to one another in many cases of tuberculous peritonitis, and the difficulty of exposing the lumen of the intestines which results from the adhesion, this omission is not surprising. Judging from the cases in which ulceration is mentioned, we may say that it was probably present in about half of the number. It may be mentioned, however, that the ulceration was by no means always extensive. In three cases only a single ulcer was present, and in other instances it is mentioned that the ulcers showed signs of healing. In one case an ulcer is said to have completely healed.

Another important question, perhaps from the point of view of prognosis, is the frequency of pulmonary lesions, and here it is necessary to remember that I included only those cases in which, so far as I was able to judge from the brief clinical abstracts which accompanied the post-

mortem records, the disease during life had been considered to be abdominal. In 17 cases the lungs appear to have been quite free from disease, and in 7 other cases the disease was limited to the pleuræ, the lungs themselves being healthy. In 21 the disease present was of recent date, and in most instances took the form of scattered tubercles. In the remaining 35 cases, that is in rather less than half, there were older lesions, such as cavities, which were generally small, or localised fibro-caseous disease was present.

Since intestinal ulceration is so commonly present in cases dying of tuberculous lung disease, I thought it would be interesting to see whether in these cases intestinal ulceration appeared to be dependent upon old pulmonary lesions, that is to say whether it could have been set up by the swallowing of infected sputum. I cannot say, however, that any connection between the two was very evident. In 13 cases of intestinal ulceration the disease of the lungs was of some standing, but in 9 cases the lungs appeared to have been perfectly healthy, and in 7 the disease was quite recent.

A brief reference may be made to the female generative organs. In the Guy's Hospital records only, perhaps, was there evidence that they had been systematically examined. Fourteen of the Guy's Hospital cases were in females and of these 14 there was tuberculous disease of the Fallopian tubes in 5; but two of these cases occurred under the age of 1 year and one under 2 years, another occurred at the age of 9, and the fifth only was in an adult aged 28 years. In the Westminster Hospital records similar disease of the Fallopian tubes is mentioned in a woman aged 25, and in another aged 40, and in the Bristol Royal Infirmary records, one occurred in a girl aged 19, and another in a woman aged 23. Disease of the Fallopian tubes is probably more common than these records show, but since tuberculous peritonitis is as common in the male as in the female sex, it seems improbable that the female generative organs are often the starting point

of the disease. These 80 cases were about equally divided between the two sexes, 41 were in females, 39 in males.

The period of life in which the disease was most common was the first five years. Twenty-two, more than a quarter of the number, occurred at that early period; but this probably does not represent the comparative frequency of the disease in very early life, since these statistics were taken from general hospitals where the percentage of young children is not large. Thirty-two cases occurred between the ages of 5 and 20 making, with the cases which occurred before the age of 5, 54 cases in the first 20 years, that is to say about two-thirds of the cases were in the first two decades of life. Cases occurred, however, at almost any age the oldest being 67.

In conclusion, it may be said that the estimation of the frequency of permanent recovery in tuberculous peritonitis appears to be a difficult task in the post-mortem room. Adhesions evidently of tuberculous origin are sometimes met with in patients who have died of some non-tuberculous disease, but they are generally localised to a very small area of the abdomen. If, however, one can argue from comparison with the pleura one may say there is no reason why recovery should not be complete and lasting. In my own experience, in about 90 per cent. of cases of general fibroid adhesion of the pleura found in patients dying of various diseases after middle life there has been distinct evidence that the pleurisy which gave rise to the adhesions had been tuberculous.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902).

19. TUBERCULOUS PERITONITIS: PATHOLOGY AND MORBID ANATOMY.

By WAYLAND C. CHAFFEY, M.D.

The tables appended are compiled from abstracts of records which formed the basis of a thesis for the degree of M.D. of London University in 1889, together with an appendix of further work on the same lines, and published in 1890. At that time they interested me chiefly on account of certain views as to the causation of tuberculosis. I now have recourse to the same series of cases for the purpose of demonstrating the relationship of tubercle of the peritoneum to other lesions of a tuberculous nature, and I hope that some at least of the conclusions to be drawn therefrom may prove of interest.

Examples of some of the conclusions which may be be drawn, after perusing the foregoing tables.

That peritoneal tubercle was present in 28 per cent. of the 118 cases of tuberculosis examined post-mortem.

That peritoneal tubercle without ulcers of the bowel amounted to 10 per cent. of all cases, or 27 per cent. of tubercle of the peritoneum.

The chronic tubercle without ulcers of the bowel amounted to $7\frac{1}{2}$ per cent. of all cases, or 20 per cent. of tubercle of the peritoneum.

That recent tubercle without ulcers of the bowel amounted to $2\frac{1}{2}$ per cent. of all cases, or 7 per cent. of tubercle of the peritoneum.

That peritoneal tubercle without ulcers amounted to 28 per cent. of all cases, or 73 per cent. of cases in which tubercle was found in the peritoneum, viz. 29 per cent. of cases in which chronic tubercle was found in the peritoneum, and 44 per cent. of cases in which recent tubercle was found.

That the mesenteric glands were diseased in all the

cases of tuberculous ulceration of the bowels, and 42 per cent. of the cases of tubercle of the peritoneum occurred without ulcers of the bowels.

That the mesenteric glands were diseased and tuberculous in 50 per cent. of all cases of tuberculosis. In one case only were they the sole seat of tuberculosis. In 80 per cent. of all cases the oldest lesion appeared to be in the bronchial glands, whilst in 20 per cent. the abdominal lesions appeared to be older than elsewhere.

Further it will be found—

That peritoneal adhesions, more or less extensive, were present in 58 per cent. of the cases of peritoneal tubercle.

That the lungs were tuberculous in all the cases of recent tubercle of the peritoneum, and in 77 per cent. of the cases of chronic tubercle of the peritoneum.

In one case of peritoneal tubercle no other tuberculous lesion was discovered, except in the bronchial glands.

It is necessary to remember that these relations can only be held to subsist post-mortem, the conditions of organs being different in cases not far advanced towards a fatal termination. This difference may, in some cases, be one of degree rather than of extent.

Whilst engaged in the work of determining the incidence of tuberculous ulceration of the intestines, I was met with the difficulty of deciding, in some few instances, what was and what was not fairly to be included in this category and I elected to classify as tuberculous only such ulcers as presented definite tubercles to the naked eye. It is, probably, by the exclusion of such other cases of doubtful nature that my statistics present somewhat lower figures in this connection than those given by my old friend and former chief, Dr. Ashby, and by others who have made valuable contributions to the literature of the subject.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902.)

An analysis of 118 *consecutive cases of tuberculosis, in which post-mortem examinations were made by Dr. Chaffey at the Children's Hospital, Great Ormond Street, London, and at the Children's Hospital, Brighton.*

TABLE I.—Showing the relationship of tubercle of the peritoneum to tuberculous ulceration of the intestines and to tuberculous disease of the mesenteric glands.

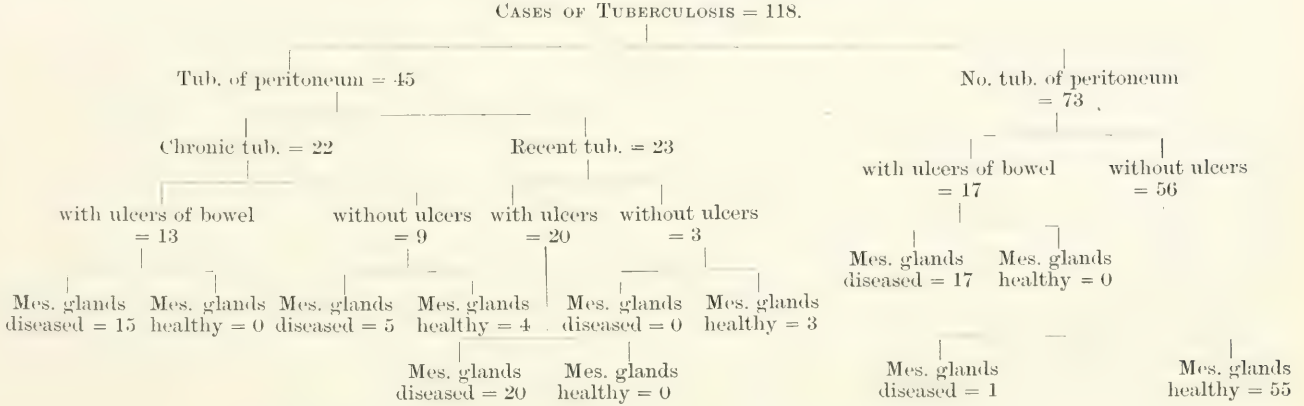
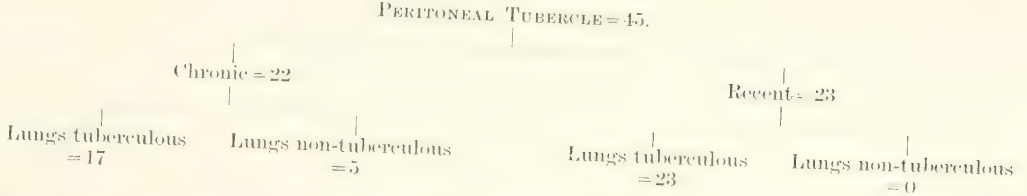

CASES OF TUBERCULOSIS = 118.
Tub. of peritoneum = 45
No. tub. of peritoneum = 73
Chronic tub. = 22
Recent tub. = 23
with ulcers of bowel = 13
without ulcers = 9
with ulcers = 20
without ulcers = 3
with ulcers of bowel = 17
without ulcers = 56
Mes. glands diseased = 15
Mes. glands healthy = 0
Mes. glands diseased = 5
Mes. glands healthy = 4
Mes. glands diseased = 0
Mes. glands healthy = 3
Mes. glands diseased = 17
Mes. glands healthy = 0
Mes. glands diseased = 20
Mes. glands healthy = 0
Mes. glands diseased = 1
Mes. glands healthy = 55

Table II.—Showing the relationship of tubercle of the peritoneum to adhesions of the peritoneum, and to tuberculous ulceration of the intestines, among the 45 cases of peritoneal tubercle.

Tubercle;	adhesions;	no ulcers = 10 (tub. chronic in 8)	} 26, adhesions.	
Tubercle;	adhesions;	ulcers = 16 (tub. chronic in 9)		
Tubercle;	no adhesions;	ulcers = 17 (tub. chronic in 4)	} 19, no adhesions.	
Tubercle;	no adhesions;	no ulcers = 2 (tub. chronic in 1)		

Total tubercle of peritoneum = 45

Table III.- Showing the relationship of peritoneal tubercle to pulmonary tubercle.



Peritoneal tubercle = 45 (about 38 per cent. of all cases of tuberculosis).

Adhesions with peritoneal tubercle = 26 (about 22 per cent. of all cases of tuberculosis, and 58 per cent. of peritoneal tubercle).

Av. age of cases of peritoneal tubercle = $5\frac{1}{2}$ years.

In 35 cases of peritoneal tubercle the ages ranged from 9 months to 9 years; whilst in 10 the ages ranged from 10 years to 14 years. In the former division the tubercle was chronic in 43 per cent.; in the latter division the tubercle was chronic in 70 per cent.

20. TUBERCULOUS PERITONITIS: PATHOLOGY AND MORBID ANATOMY.

By GEORGE CARPENTER, M.D.

Of 60 cases of abdominal tuberculosis which were examined by me in the post-mortem room of the Evelina Hospital for children 32 occurred in males and 28 in females. Their ages were as follows. During the first six months of life 1 case; second six months, 2 cases; at 12 months 6 cases; 18 months 1 case; 2 years 10 cases; 3 years 10 cases; 4 years 7 cases; 5 years 5 cases; 6 years 3 cases; 7 years 2 cases; 8 years 5 cases; 9 years 2 cases; 10 years 2 cases; 11 years 1 case; 12 years 1 case; and in two instances the ages of the children were not recorded. Total 60 cases.

Of the above number 25 children came under treatment for abdominal symptoms and I propose to deal with them first.

An analysis of these 25 fatal cases shows that no less than 17 of them had obvious enlarged and caseating mesenteric glands. In one instance these glands were enlarged and of stony hardness, in another they were enlarged and fibrous. In 12 cases these enlarged and tuberculous mesenteric glands were associated with intestinal ulceration; but in 5 there was no ulceration, and in two of these latter instances there was some injection of Peyer's patches and of the mucous membrane of the cæcum.

In 13 of these 17 cases there was associated peritonitis. Of these 13, 3 showed recent lymph, in 1 there was a perforation, and in 1 a large abdominal abscess cavity. In the 8 cases remaining of the 13, in 6 of them there was matting of the bowels the adhesions displaying caseous granulations, or caseous masses, or grey granulations; and in 1 instance tubercles were not visible in the lymph; and in

1 case there were dense adhesions everywhere, apparently free from tubercles, associated with stinking pus.

Of the 7 remaining cases, in 4 the abdominal cavity was obliterated and it was impossible to say what anatomical structures were involved on inspection of the agglutinated masses of lymph, cheesy material, tubercles and intestinal remnants. In one of them no obvious tubercle could be detected. There was one case of uncomplicated peritonitis in a child 4 months old. Its intestines were covered with a diphtheritic-membrane-like deposit which could be readily peeled off and left a polished surface; this was crowded with tubercle bacilli. In one case the serous membranes generally (pericardium, pleuræ and peritoneum) were involved, together with a few scattered tubercles in the lungs. The last was that of a child who had recovered some time previously from peritonitis, but was re-admitted into the Evelina Hospital with a very large empyema and died during the operation undertaken for its relief. At the autopsy some few trifling abdominal adhesions were found, together with two grey granulations on the peritoneal surface of the abdominal wall, but no tubercle visible to the naked eye could be seen elsewhere.

It would appear from this that the mesenteric glands are common starting points of the disorder; that intestinal ulceration is by no means a necessary preliminary to the production of tuberculous mesenteric glands, but that, although that is so, intestinal ulceration and caseating mesenteric glands are very frequently found associated. Further, that peritonitis although not a necessary sequence of the preceding condition perhaps not infrequently accompanies it. Also, that tuberculous peritonitis may be present in marked degree without the intestinal follicles or mesenteric glands being implicated. Lastly, intestinal ulceration is almost invariably accompanied by glandular disease, but, on the other hand, the glands may at times be far advanced in disease with an apparently sound mucous membrane.

Of these 25 cases of abdominal tuberculosis, 4 were limited to the mesenteric glands, intestines and peritoneum, and 1 was strictly localised to the peritoneum.

There were 9 cases of mixed glandular, intestinal and peritoneal infection which were not so limited, but were associated with tuberculous lesions elsewhere of a purely secondary character. Of these, 8 had their lungs implicated in varying degrees. In 6 of these 9 cases aforesaid the pleura was involved. Of these 6 cases 3 had tuberculous bronchial glands likewise, and 1 of these 3 died of tuberculous meningitis. In 1 case the spleen and kidneys were also affected, and in 1 the spleen and liver.

Two cases of tuberculous mesenteric glands (one with recent peritonitis) were associated with tubercles in the lungs; in one instance with the liver in addition and in the other with calcified bronchial glands, both cases being undoubtedly secondary to the abdominal trouble.

One case of intestinal ulceration and enlarged mesenteric glands was associated secondarily with miliary tubercles in the lungs, peritoneum, liver, spleen, kidneys, and fundus oculi.

One case of caseous mesenteric glands together with a thickened omentum and matted intestines developed miliary tubercles in the lungs, liver, spleen, and kidneys.

One case of caseating mesenteric glands together with nodules in the peritoneum and a large abscess cavity was associated secondarily with a few scattered tubercles in one of the lungs, and a trifling collection of pus in one pleura.

One case of partially healed intestinal ulcers in the ascending colon, the mesenteric glands being free from obvious disease, was found to have old pleurisy over the apex of one lung in which a solitary tubercle was seen planted, together with an abscess in the right half of the cerebellum.

One case whose intestines were matted together into a solid mass with tubercles developed miliary tubercles in the lungs, and the bronchial glands were large and cheesy.

Of the remainder, 4 cases were associated with tuberculous lesions elsewhere; 1 had a few tubercles in the upper part of the lower lobe of one lung; 1 had a tuberculous cavity, the size of a walnut, and old adhesions at one lung apex; 1 was associated with tuberculous inflammation of the serous cavities, together with a few tubercles in the lungs; and 1 was associated with an empyema.

Implications of other organs in these fatal cases was therefore not uncommon, and of other organs the lungs were most often attacked, viz.:—19 out of 25 cases.

Reviewing the 35 cases, temporarily left over, I find that 22 of them came under medical care for lung affections, one of their number developing tuberculous meningitis; that 9 were attended for cerebral symptoms, 8 being meningitis and 1 a cerebellar tumour. In one of the meningitis cases tuberculous nodules were found in the cerebellum and the seminal vesicles were also tuberculous. One case attended with typhoid symptoms, 1 was primarily abdominal and developed meningitis and a tubercle in the choroid; in 1 case definite localising symptoms were wanting; and 1 was brought by its mother for marasmus.

Of these 35 cases, in 20 the initial lesions found post-mortem were confined to the lungs and bronchial glands; in 2 cases it was not certain whether the cheesy mesenteric glands or the lungs were the primary seat; and in 6 lung cases, not counted with the above, no definite statement was made in their notes about the bronchial glands. Three cases were probably primarily intestino-glandular, and 1 case was undoubtedly so. One doubtfully primarily intestinal developed meningitis and a choroidal tubercle; and 1 other similar case had caseous Fallopian tubes of endogenous origin, cerebellar and pontine tuberculous tumours, with optic atrophy and choroidal tubercles at the yellow-spot regions of the fundi oculorum.

Further, of these 35 cases no less than 26 were affected with either tuberculous mesenteric glands, tuberculous intestinal ulcers, or a combination of the two. In 9 cases the glands were affected without intestinal ulceration; in

2 of these the intestinal mucous membrane was injected, in 1 there was injection and some tubercles were visible in the mucous membrane, in 1 there was a tuberculous deposit the size of a florin in the mucous membrane of the colon. In 1 case there were intestinal ulcers and the mesenteric glands were not affected to the naked eye.

In regard to peritonitis flakes of lymph were found in 1 case only, but there were several cases with adhesions round the liver and spleen. In 3 cases there was serous fluid in the peritoneal cavity, 1 of them being associated with tubercles. In 3 cases there were miliary tubercles in the peritoneum, without fluid or peritonitis.

Of the 35 cases, in no less than 33 the lungs were affected, and in 20 of them the pleuræ were involved in varying degrees. In 7 cases the lungs were attacked without implication of the intestino-glandular system, and in 2 cases the mesenteric glands were involved apart from the lungs. It is evident, therefore, that the lungs rarely escape in fatal cases of tuberculosis of the intestino-glandular system, and, conversely, in the majority of instances tuberculosis of the lungs is associated with tuberculosis of the intestino-glandular system.

Excluding the lungs, of these 35 cases 27 were associated with tubercles in organs other than in the capsules of the liver and spleen and in the peritoneum. In 6 of the cases the other organs were not attacked. In 1 case there was meningitis, and in 1 case a post-pharangeal abscess. It appears that doubts as to the identity of bovine and human tuberculosis cannot be substantiated inasmuch as human tuberculosis can be experimentally transmitted in cattle. Both diseases must be identical otherwise it is necessary to assume that the human donor at the time of the experiment was suffering from bovine tuberculosis. On the other hand, Koch has not been able to find after a prolonged inquiry a case of transmission of abdominal tuberculosis from the milk of cows suffering from perlsucht. There is no doubt that topical vulnerability of the individual comes largely into play in cases of abdominal tuberculosis as well

as in other tuberculous affections. Thus, the prolonged swallowing of the tuberculous sputum in children is followed in only a certain proportion of cases by intestinal tuberculosis. Again, tuberculosis starting in the digestive tract is comparatively uncommon on the one hand and on the other the opportunities of contracting this disease from tuberculous foods are numerous.

Summary.

1. Tuberculous disease originates in some children in the intestines or abdominal lymph glands. Either of these structures, and less commonly the latter, or both in combination may be involved. These lesions are mostly associated with peritonitis.

2. Cases that are strictly confined to the intestines, the mesenteric glands and the peritoneum are comparatively rare ; to the peritoneum alone, or combined with tuberculous inflammation of the serous membranes rarer still.

3. Implication of other organs in fatal cases of primary abdominal tuberculosis is not uncommon and of all organs and structures the lungs appear to be most likely to be attacked. Invasion of the lungs in such cases probably occurs *via* the thoracic duct and pulmonary artery, the lungs acting as a filter. This is contrary to the experience of others who consider that food tuberculosis may get well or spread but never leads to tuberculosis of the lungs.

4. Primary abdominal tuberculosis is sometimes associated with tuberculosis of other parts which are free from external entrances, *e. g.* the brain, the meninges, the pericardium, the pleura, the choroid, etc.

5. The majority of cases of ulcerating tuberculosis of the lungs contract tuberculous intestinal ulcers, tuberculous lymph nodes or a combination of the two conditions. This mode of onset of abdominal tuberculosis is more common than the other. It is not so frequently associated with peritonitis. The clinical symptoms in such cases are either pulmonary or perhaps cerebral and not often abdominal.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902.)

21. TUBERCULOUS PERITONITIS: PROGNOSIS.

By G. A. SUTHERLAND, M.D.

The prognosis in tuberculous peritonitis is a subject on which many and diverse views have been expressed. The great question as to whether the disease tends to run a favourable or unfavourable course may be said to be still unsettled. Twenty-five years ago the prognosis was regarded as hopeless. Ten years ago it was considered more hopeful, especially if surgically treated. To-day we are still searching for more light, and although extensive statistics have been compiled on the Continent, which have considerably influenced opinion in this country, there are comparatively few published from English hospitals.

I propose to limit my remarks on prognosis to the results of a study of 41 cases of tuberculous peritonitis treated as in-patients at Paddington Green Children's Hospital. These represent all the cases of that affection which are available for statistical purposes from that hospital, a few having been rejected because the diagnosis was uncertain or the period under observation was too brief.

The first important point in the prognosis is to ascertain the proportion of recoveries to deaths. In these 41 cases there are—

Recoveries	.	29, or 70·7	per cent.
Unrelieved	.	1 ,, 2·5	,,
Deaths	.	11 ,, 26·8	,,

If the results of medical treatment are compared with those of surgical treatment, the statistics are—

Medically treated 27 cases.	Recovered	22, or 81·3	per cent.
	Unrelieved	1 ,, 3·7	,,
	Died .	4 ,, 15	,,
Surgically treated 14 cases.	Recovered	7 ,, 50	,,
	Died .	7 ,, 50	,,

In the case of a chronic affection like tuberculous peritonitis it can hardly be deemed correct to use the term

"recovery" when the patient leaves the hospital or convalescent home; "apparently on the road to recovery" would be as far as one can safely go. The cases therefore have been followed up as well as possible after leaving the hospital, and the statistical results, both favourable and fatal, are drawn up after keeping the patients under observation for the longest possible time. The cases which are entered as "recovered" were under observation for the following periods.

Under 3 months	2
From 3 to 6 months	7
„ 6 „ 12 „	1
„ 1 „ 2 years	4
„ 2 „ 3 „	4
„ 3 „ 4 „	1
„ 4 „ 5 „	4
„ 5 „ 6 „	3
Over 6 „	3
Total	29

Considering the number of cases which were under observation for a year or longer—namely, 19, or 66·5 per cent.—it will perhaps be admitted that the term recovery is justified. Further, in those cases the signs of active disease had ceased, the traces of local disease became less evident with each succeeding year they were observed, and the general strength seemed steadily to improve.

The natural course of tuberculous peritonitis.—Tuberculous peritonitis in its uncomplicated form is an affection which lasts usually for from six months to a year, or longer. The natural course of the disease in a fairly healthy child would appear to be towards ultimate recovery. Perhaps in no form of tuberculosis does nature make greater or more successful efforts to cope with the disease. The formation of fibrous tissue is nature's method of shutting up the tubercles, cutting off their supply of nourishment, and starving them out. When their vitality is gone and all active peritoneal tuberculosis has ceased,

nature next directs the powers of the body to the absorption of the dead tubercles and the fibrous tissue surrounding them. Sometimes the effort is completely successful, sometimes the absorption is only partially complete, and sometimes complications arise from outside causes which put an end to the process. If this process of fibrosis is to be regarded as the natural course of the illness towards a favourable issue, the prognosis will depend on the patient's powers of furthering it. Consequently, a strong family history of tuberculosis, or an infancy passed under bad hygienic and dietetic conditions, or a constitution of slight resistent power, or a history of one or more severe attacks of infective fever during early life must be regarded as factors which influence unfavourably the prognosis in any given case.

The prognosis in uncomplicated tuberculous peritonitis.—The disease presents itself under various clinical aspects. There may be an acute onset with typhoid-like symptoms, followed by a very severe illness, characterised by ascites, abdominal distension, diarrhœa or constipation, and high temperature. Although one would hesitate to speak decidedly during the acute stage of the illness it is not uncommon for such cases to terminate in complete recovery. Most of the cases begin insidiously. The ascitic type, in which fluid is poured out more or less freely owing to the irritation produced by many scattered tubercles, is one in which the prognosis is favourable if it is succeeded by a fibro-plastic formation, which is the natural termination. Those cases in which the fibro-plastic formation is active from the start do well as a rule. Cases of tumour formation, rolled up omentum, or puckered up mesentery, or matted intestine, may present no active symptoms, are extremely chronic, and usually terminate favourably. A common clinical type is that in which tuberculous pleurisy is an associated condition, and the prognosis does not appear to be unfavourably influenced by this association.

Various symptoms in the course of the disease affect the prognosis. A favourable condition may be said to be pre-

sent when the *temperature* is subnormal, or normal, or only slightly raised, and even when there is a considerable evening rise to 102° F. or 103° F., it is wonderful how well and how long the patient will hold his own, provided there is a daily remission and apyrexial period. On the other hand, continuous pyrexia, even of moderate degree, is unfavourable, is usually accompanied by progressive loss of strength, and is suggestive of some complication. A continuously rapid pulse, over 110 per minute, is to be regarded as indicative of acute abdominal disease, and is of unfavourable prognostic significance. Diarrhœa when prolonged is of bad omen, and suggests one of the gravest complications, tuberculous ulceration of the bowel. Great *distension of the stomach and intestines*, when paralytic in origin, is of unfavourable significance; when it is obstructive in origin it may be amenable to surgical treatment. *Rapid wasting* must be looked on as an indication that the disease has gained the upper hand locally or is becoming generalised. The defensive powers of the constitution are invariably weakened in the presence of rapid emaciation. *Recurrent attacks*, with or without marked pyrexia, affect the prognosis unfavourably. They tend to exhaust the resistent powers of the patient, and they point to fresh infection from an active although possibly unrecognised source.

The prognosis in complicated tuberculous peritonitis.—The complications which may precede or arise from tuberculous peritonitis seriously affect the prognosis, and one or more of them will usually be found to be present in the fatal cases.

1. Tuberculous ulceration of the intestine. This may be small in extent, and not associated with any marked symptoms. When it is extensive, as indicated by intractable diarrhœa, and the passage of blood and mucus, the prognosis is unfavourable.

2. If extensive caseation of the mesenteric glands or of tuberculous masses exists, recovery is delayed, fresh infection of the peritoneum is apt to occur, and the risk of abscess formation and rupture is constantly present.

3. Localised suppuration, from infection through glands or the intestine, is a complication which, if unrelieved by surgical treatment, is apt to retard recovery and lead to still more serious complications.

4. Obstructive symptoms from intestinal matting or the pressure of tuberculous growths are as a rule not marked. If progressive and unrelieved, the prognosis is bad, but surgical interference may prove successful in removing all imminent danger.

5. In the presence of pulmonary tuberculosis, with definite physical signs, the prognosis is unfavourable. It must be remembered, however, that dulness and crepitations are frequently present at one or both bases, owing to the diaphragm being pushed upwards, the lower portions of the lungs being thrown out of action, and congestion occurring in consequence. With the relief of the abdominal distension, these pulmonary signs will usually disappear entirely. The prognosis is very bad in the case of the following complications.

6. The rupture of a glandular or other abscess, or the perforation of an intestinal ulcer into the general peritoneal cavity.

7. The onset of tuberculous meningitis.

8. The occurrence of general miliary tuberculosis.

As confirming the serious prognostic influence of the various complications the causes of death in the fatal cases may be given here. Six of the eleven cases were examined post mortem.

Purulent peritonitis from intestinal perforation or ruptured abscess	5 cases
Intractable diarrhœa (? intestinal ulceration)	2 „
With cerebral symptoms (? tuberculous meningitis)	2 „
Tuberculosis of peritoneum, pleura, and lung, with partial intestinal obstruction by a band	1 case
Cause unknown	1 „
Total	11 cases

The prognosis as affected by medical treatment.—The results of medical treatment as given above may be

regarded as favourable. If the prognosis in uncomplicated tuberculous peritonitis depends on improving as much as possible the patient's resistent powers until the tuberculous process is checked, and increasing his aggressive powers until the tuberculous products have been entirely removed, it is plain that medical treatment directed to these ends will be of the highest importance in making the prognosis more favourable. In the complicated forms of the disease medical treatment is of less direct value.

The prognosis as affected by surgical treatment.—In the complicated forms of tuberculous peritonitis the prognosis is much more serious, and here it is that the prospect may be entirely changed by surgical interference. One striking case is before me, that of a girl of three years, on whom Mr. Watson Cheyne performed laparotomy for obstruction, and short-circuited the small intestine. The patient made an excellent recovery. In another case, with suppurative peritonitis from a perforation of the cæcum, a fatal termination ensued.

As regards the surgical treatment by simple laparotomy —the so-called cure for tuberculous peritonitis—our results are not good, namely, six recoveries and six deaths. It is not suggested that the fatal results were due to the laparotomy, for the deaths occurred at periods of from one to six months after the operation. But it is clear that the operation was ineffectual in preventing the complications which lead to a fatal termination. The cases which recovered may be said to have done so in spite of the operation, for they presented in their post-operative stages no striking changes and no clinical developments which could not be equally well observed in the cases treated medically. At one time I was a supporter of laparotomy as a routine treatment, but further experience has altered my opinion. A girl of six years, the subject of fibro-ascitic tuberculous peritonitis, was considered a suitable case for laparotomy, but operation was deferred for unavoidable reasons, and she was sent to the country. While she was there the abdominal contents became harder and denser,

until they felt like plaster of Paris, the fluid disappeared, and the girl improved. Some months later she was so much better that operation was not considered necessary, and during the next year there took place a gradual absorption of all the dense material, until finally she was quite restored to health and the abdomen to a normal condition. In other cases before me, cases of acute tuberculous peritonitis, of tumour formation, of the ascitic type, and of the fibrous variety, I find that as good results may follow from medical as from surgical treatment, and I do not find that the course of the disease is appreciably affected by laparotomy. From the post-mortem evidence also it is plain that, whether the case was medically or surgically treated, death was not due to uncomplicated tuberculous peritonitis, but to some complication beyond relief either by medical measures or by laparotomy.

The conclusions drawn above may be summarised as follows:

1. In uncomplicated tuberculous peritonitis the prognosis is good.

2. When tuberculous pleurisy is present the prognosis is still favourable.

3. The prognosis is rendered less favourable in the case of (*a*) a strong family history of tuberculosis, (*b*) an infancy passed under bad hygienic and dietetic conditions, (*c*) a constitution of feeble resistent power, or (*d*) a history of severe infective illness in early life.

4. The prognosis is rendered less favourable in the presence of one or more of the following symptoms. Continuous pyrexia, rapid wasting, persistent diarrhœa, rapid pulse, and recurrent acute exacerbations.

5. The prognosis is rendered less favourable in the presence of one or more of the following local complications:—(*a*) tuberculous ulceration of the bowel, (*b*) extensive caseation of the mesenteric glands or of tuberculous masses, (*c*) localised suppuration from infection through glands or the intestine, and (*d*) obstructive symptoms from bands or matting in the intestine.

6. The prognosis is bad in the case of the following complications:—(*a*) the rupture of a suppurating gland or the perforation of an intestinal ulcer into the peritoneal cavity, (*b*) pulmonary tuberculosis, (*c*) tuberculous meningitis, and (*d*) general miliary tuberculosis.

7. In tuberculous peritonitis the prognosis is not appreciably affected by simple laparotomy.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902.)

22. TUBERCULOUS PERITONITIS: MEDICAL TREATMENT.

By LEONARD GUTHRIE, M.D.

In discussing the medical treatment of tuberculous peritonitis I am hampered by the prevalent notion that surgeons alone are able to cure this disease.

Mr. Watson Cheyne, for instance (Harveian Lectures, 1899, p. 59), asserts that "the prognosis of tuberculous peritonitis, when no surgical intervention has taken place, is undoubtedly very grave, but much depends on the form of the disease and on the presence or absence of complications." With the latter part of the sentence I agree, but to the former I venture to demur.

It implies that the prognosis when surgical intervention has taken place is no longer very grave, and that those who do not call in the aid of a surgeon are guilty of allowing a very grave prospect to continue when it might easily be converted into one more favourable.

If Mr. Cheyne's statement be accepted literally, any discussion on methods of treatment other than surgical would be a waste of time. But, my own experience of tuberculous peritonitis leads me to think that the prognosis

in uncomplicated cases in children is by no means so grave as we are taught to believe, and that the triumphs of surgery in curing this disease have been somewhat over-rated.

Of 41 cases treated at the Paddington Green Children's Hospital, 14 underwent laparotomy and 7 of these died, whilst of the remaining 27 treated medically, only 4 were fatal.

The death rate in surgical cases was therefore 50 per cent., whilst in medical cases it was under 16 per cent. Moreover, in the 4 fatalities under medical treatment, the cause of death in three was septic peritonitis due to perforation or rupture of a mesenteric glandular abscess; in one, death was due to extensive pulmonary phthisis, also present in two of the others. All of them died within a month of entering the hospital, and had they undergone laparotomy they would only have increased the percentage of surgical failures.

I am prepared to be told that the surgical cases were more severe than the medical which recovered, and admit the fact so far as the fatal surgical cases are concerned, but not as regards those which survived laparotomy.

It has also been urged that cases which recover under medical treatment are not suffering from tuberculous peritonitis at all. Yet it must be remembered that the diagnosis is in practically all cases made by the physician, and it seems a little hard that he should only be right when he calls in a surgeon and always wrong when he does not.

Before discussing further the claims of surgical over medical treatment, I must follow the programme and deal with the latter.

Medicinal treatment.—We cannot claim at present to slay the tubercle bacillus within the body by any drugs, but the resolvent action of such drugs as mercury and iodide of potassium on tuberculous inflammations is open to discussion. Most of the medical cases were treated by mercurial inunction or by biniodide of mercury given in-

ternally, and recovered, whether in consequence of the treatment or not is a matter of opinion. Some got well without mercurial treatment.

Arsenic.—Is arsenic of use in tuberculous affections? It is believed to cure some cases of lymphadenoma, and it is now well known that so-called lymphadenoma in children is frequently tuberculous. It would, therefore, be interesting to discover whether these are the cases in which arsenic is of value; for if so, arsenic might affect the course of tuberculous peritonitis. It is used extensively in America, I believe, with this object.

I know no other drugs which can conceivably have influence over tuberculosis, but by some, guaiacol carbonate and iodoform are held in repute. Cod-liver oil, of course, is used systematically, but probably as food rather than medicine. Syrup of iodide of iron has a reputation, but I doubt if it is superior to other preparations of iron when anæmia is present.

Medicinal treatment of tuberculous peritonitis is almost entirely symptomatic.

The chief symptoms which call for treatment are pain and abdominal tenderness, flatulency and indigestion, diarrhœa or constipation, vomiting and ascites.

Pain is seldom marked in patients kept at rest in bed unless it is due to the onset of acute septic peritonitis.

The ordinary slight pains and tenderness can usually be relieved by local applications of belladonna, heated cotton wool or spongiopiline, or in some cases by painting with iodine. Opium is seldom required.

Flatulency and indigestion need careful dieting and bismuth.

Diarrhœa unless profuse and continuous needs no active treatment. In some cases it may even be salutary in reducing ascites. Bismuth and opium or aromatic chalk and opium powder are most useful remedies. Coto and various other astringents seem of doubtful utility.

With the exception of biniodide of mercury I have little faith in so-called intestinal disinfectants. They seem

to be more useful when added to the evacuations than when taken internally.

Diarrhœa when dependent on intestinal ulceration is most difficult to control. If the ulcers are situated low down in the bowel, irrigation and astringent or soothing enemata may be of service, but when situated high up such treatment is of little use.

Constipation.—A loaded rectum should be cleared out by enemata rather than by purgatives, but an occasional dose of castor oil, cascara, or pulvis glycyrrhizæ compositus may be given, unless evidence of ulceration in the shape of blood-stained or black offensive and purulent stools is present. It must be remembered, however, that ulceration may exist without any such evidence.

Medical Treatment of Ascites.

It is a question for discussion whether in cases of ascites with constipation and scantiness of urine, but without evidence of ulceration, purgatives and diuretics are indicated.

I once treated such a case in this manner for a considerable time and the patient recovered. But this treatment is hardly to be recommended as a routine process.

Vomiting is seldom excessive in children at rest and properly fed, unless signs of intestinal obstruction are present, which call for immediate surgical interference. When due to gastric disturbance only, bismuth and hydrocyanic acid are the best remedies.

Diet.—It is seldom necessary to feed a patient on fluids alone unless there is obviously extensive ulceration. The pyrexia present is rarely high enough to interfere with digestion, and harm may be done by restricting patients to the diet usual in enteric fever.

One must, of course, keep indigestible substances out of the dietary. In one case fever, vomiting, and dangerous colic following a diet including green apples, first drew attention to the existence of tuberculous peritonitis.

Starchy and other foods which in excess tend to induce flatulency and fermentation must be given sparingly.

Meat, fish, and poultry can usually be assimilated if carefully minced and prepared. Eggs, cream, milk and butter may be freely supplied. In some cases of intractable diarrhœa a diet of raw or partially cooked meat seems efficacious. When stimulants are required, brandy, if it can be procured, or old dry port from the wood are the best.

Hygiene.—In every case rest in bed, at all events in early stages, is essential, and it should be in country open air.

Sometimes children have been sent into the country merely in the hope of recruiting them sufficiently to undergo laparotomy, but in a few weeks they have returned practically restored to health. Also in most of the cases which recovered after laparotomy improvement was not manifest until the effect of country air was tried.

Hence, I believe that open-air treatment is even more eminently suitable to cases of tuberculous peritonitis than to those of pulmonary phthisis, and I would send all cases to undergo it under careful supervision and nursing, as soon as the diagnosis has been made or even suspected.

Rest in the recumbent position should be maintained until all active symptoms have disappeared.

Medical treatment then simply consists in keeping the patient at rest, in supplying a nutritious fattening and abundant diet, in the relief of incidental symptoms as they may arise, and, above all, in securing the advantage of open country air.

Radical Treatment of Ascites.

The next question which arises is when should fluid be left in, and when should it be let out?

In all varieties of cases it may subside spontaneously after rest in bed and mercurial inunction, but it also comes and goes whether mercury is used or not. In acute cases unless fluid produces great distension and distress it is

inadvisable to interfere, for if removed it will speedily reaccumulate. Surgeons have discovered that laparotomy is seldom of use in such cases. But, as in some cases of pleural effusion the withdrawal of a few ounces of fluid is sometimes sufficient to cause absorption of the rest. The supposed danger of setting up general tuberculosis by absorption of the fluid into the circulation is, I think, unfounded.

General tuberculosis is far more likely to be due to carriage of bacilli by the circulation from tuberculous caseating glands to distant parts.

In more chronic cases, when fluid shows no tendency to subside it should be withdrawn.

Tapping or aspiration in such cases, unless one is certain that the fluid is free, is dangerous. It is better to make a small incision and insert a blunt perforated trocar than to plunge a sharp instrument through the abdominal wall at the risk of wounding adherent intestines.

It has been said that "simple puncture alone is quite ineffectual." But four cases in which the fluid was free and excessive recovered after being so treated, and in only one of these was it found necessary to repeat the process, and then only after the lapse of one year. It is, however, sometimes ineffectual, and the case should then be treated by simple incision or laparotomy. This is the operation which is supposed to have revolutionised the prognosis in tuberculous peritonitis.

It is of the simplest character. The surgeon makes a two-inch incision through the abdominal wall, lets out any fluid which may be present, calls upon the bystanders to observe the resemblance between a tuberculous peritoneum and a sago pudding, and then closes the wound. It is no longer considered necessary to irrigate the peritoneum with or without antiseptics, nor even to break down adhesions unless they are causing obstruction.

The surgeon, like an ancient Etruscan Haruspex, merely gazes at the victim's entrails, and then discovers propitious omens for the future of abdominal operations.

Many fanciful theories have been invented to explain the recovery of 50 per cent. of cases after laparotomy.

The entrance of light, air, oxygen, and of non-pathogenic organisms has been held by different theorists to effect a cure.

Mr. Watson Cheyne believes that the tuberculous fluid evacuated is replaced by anti-bacteric serum inimical to the tubercle bacilli. If this is so, a similar result should follow simple puncture, and perhaps it does, although the procedure has been regarded as quite ineffectual.

I believe, indeed, that there is no mystery whatever in the matter, but that simple laparotomy is useful only in so far as it lets out fluid. Dry cases which are also supposed to be cured by the operation would, I believe, recover equally well if let alone, provided that no complications such as extensive ulceration, septic peritonitis due to suppuration of glands or perforation, meningitis, or advanced pulmonary phthisis occur.

Indeed, the recovery of uncomplicated dry cases not operated upon compares favourably with that of laparotomy cases whose convalescence is retarded by abscesses and fistulæ arising from tuberculous infection of the operation scar. On the other hand, surgical interference is absolutely necessary in cases of obstruction by adhesion bands or stricture, and in cases where suppuration is suspected. When acute septic peritonitis from perforation or rupture of an abscess occurs an operation is the only chance, though a forlorn one, of saving life. Finally, I believe that caseous mesenteric glands should be removed, if possible, as being the chief source of fatal dissemination.

It will be observed that in all these cases something more is necessary than merely opening the abdomen and looking inside. Unfortunately, the cases in which more is necessary are those which make the mortality of the operation as high as it is; whilst the more favourable results of foreign observers are, I believe, due to operating upon cases which would get well without.

In conclusion, we owe a debt of gratitude to surgeons

who through errors of diagnosis have taught us that conditions once held to be incompatible with recovery are by no means necessarily fatal. Those who advocate indiscriminate operation may be reminded of the well-known story of Ambrose Paré, the celebrated French surgeon. Once after a battle he ran short of boiling oil, and could not sleep for thinking of the sad plight of the wounded who had not received their share. But next day he found them infinitely the better for being without it.

(*Read at the Special Meeting, for the discussion of Tuberculous Peritonitis, December the 12th,* 1902.)

23. TUBERCULOUS PERITONITIS: MEDICAL TREATMENT.

By JAMES CARMICHAEL, M.D.

I CONSIDER abdominal tuberculosis the most hopeful and curable of the tuberculous diseases of childhood. In a pathological sense it is recognised that it is not unfrequently localised in the abdomen. In 127 post-mortem examinations of abdominal tuberculosis in the Edinburgh Royal Hospital for Sick Children, 14 cases were met with in which the disease was entirely localised in the abdomen and mainly in the mesenteric glands, no tuberculous lesion being found anywhere else.

In 48 of my hospital cases there were 9 deaths. In 4 of these fatal cases abdominal section had been performed. In 3 of the cases which recovered aspiration of the ascitic fluid was undertaken. In chronic ascites it must be admitted that the tuberculous nature of the disease cannot be proved unless the abdomen has been opened, but the probability is strongly in favour of its tuberculous nature. In 13 of the cases the result "cured" is noted, *i. e.*, the patients had been kept under observation for a prolonged period, and remained in perfect health with no sign of

disease of any organs. Sixteen of the cases were dismissed from the hospital "improved." In the remaining 13 cases the records stated "*in statu quo.*" In the later years of my hospital experience cases of abdominal tuberculosis seemed to be relatively more common than other tuberculous cases, *i. e.*, general tuberculosis. I may be allowed, after a statement of these facts, in a few words to mention in general terms the treatment adopted. In recent years this has been more satisfactory than ever before in my experience, mainly, I believe, due to the greater importance attached to hygienic treatment. In all cases the children were kept in bed and in the open air as much as possible during the greater part of their hospital residence. Full diet was given according to the age of the patient, except in cases—and these were few—in which there were enteric symptoms. Cod-liver oil in one form or another was given along with food in most cases. Petroleum emulsion was given when from any cause, such as laxity of the bowels, cod-liver oil was contra-indicated. Guaiacol or beech creosote and occasionally perchloride of mercury were given in almost all the cases. Local medication, and particularly massage of the belly were attended by the most favourable results. The local unguents used were oleate of mercury with lanolin, or iodine ointment. Recently guaiacol with lanolin has been applied with apparently even better effect. With reference to massage I cannot speak too highly. I believe it is equally as efficacious, perhaps more so than laparotomy in stimulating absorption and restoring the balance of nutrition in the diseased parts. It should be used carefully twice a day for a quarter of an hour or twenty minutes, and thereafter the abdomen wrapped up with a pad of cotton wool, and a flannel binder put on firmly so as to exercise a certain amount of elastic compression.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1902.)

24. TUBERCULOUS PERITONITIS: MEDICAL TREATMENT.

By GEORGE CARPENTER, M.D.

In all the cases the usual line of treatment was adopted, and cod-liver oil, maltine, careful dieting, and rest in bed formed the chief measures; and when the child could undertake the journey it was sent to a convalescent home in the country, the latter being considered the most important of all measures. Many drugs were used, chiefly arsenic, chloride of calcium and the hypophosphites of sodium; but in several of the cases hypodermic injections of perchloride of mercury ($\frac{1}{30}$th of a grain) were administered once a day, and some benefit did appear to be derived from these administrations. This form of treatment produced cutaneous indurations, but suppuration did not occur. Local mercurial applications, in one form or another, to the abdomen were adopted in nearly all cases. The bowels were kept relieved by enemata. Abdominal pains were treated by Dover's powder in doses of 4 grains or so, three or more times daily for a child of five years, and so on in proportion, according to the amount of pain and the effect produced. My experience of abdominal section for this complaint has not been large, but from what I have seen I do not think it has any advantages over milder measures, and I have not been favourably impressed by it. Three cases of mine treated in this way developed a fæcal fistula at the cicatrices, and fæcal fistula at the umbilical cicatrix in my experience has been a rarity in children that I have medically treated. Abdominal section for the free drainage of a tuberculous abdominal abscess is, I take it, good practice; but, abdominal section for tuberculous ascites is another matter. Tuberculous ascites recovers by the older fashioned methods of rest in bed, mercurial inunction, and so forth, and inasmuch as it is not easy to see in what respect such extensive surgical treatment differs materially from the much milder

form of it in the shape of paracentesis abdominis, I prefer, when the belly is distended with fluid, to use Southey's trocar and cannula and repeat the application when necessary.

Of 54 cases treated medically, of whose subsequent history I made inquiries, 19 died, 10 are recorded as having recovered, 16 improved while under treatment, and the fate of 9 of them was not known. From this it would appear that tuberculous peritonitis is not infrequently cured, and perhaps more often resolves than any other form of tuberculosis; nevertheless, it must be looked upon as a very fatal disease. Treatment should rather be directed to preventive measures than to therapeutic, for at present there is more hope to be derived from the exercise of the former than by the use of the latter. In this respect the separation of the young from phthisical and tuberculous patients, a more efficient control of the milk and food supply, and the provision of a more healthy environment will occur to the minds of all. Unfortunately a factor of primary importance, procreation by weakly stock is not under the control of the medical profession and cannot be readily prevented.

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the* 12*th*, 1903.)

25. TUBERCULOUS PERITONITIS: SURGICAL TREATMENT.

By WATSON CHEYNE, C.B., F.R.S.

Mr. Watson Cheyne said that he must apologise for not having prepared a special paper for the meeting of the Society, but from what had already been said by the former speakers, more especially by Dr. Sutherland and Dr. Guthrie, it was well that he had not done so, because his remarks would hardly have been to the point after what had been said. He had listened with the greatest interest to the papers by Dr. Sutherland and Dr. Guthrie, the more so because he had the highest respect for their judgment.

When, therefore, they told him that they could now cure peritoneal tuberculosis without operation and that operation did no good he was very much impressed with their statement, and could not, of course, cavil at it. If their statement proved to be correct, then a mistake had been made for some years in advocating laparotomy as a general method of treatment for peritoneal tuberculosis, excluding of course cases where laparotomy in peritoneal tuberculosis was required for obstructive or other complications. If such a mistake has arisen it must be entirely attributed to physicians, and not to surgeons. If he recalled the history of the matter the Society would see that that statement was a correct one.

Years ago physicians were practically unanimous in looking on tuberculous peritonitis as a fatal disease, and not very long ago, in a leading encyclopædia of medicine, the subject of peritoneal tuberculosis, from a medical and therapeutic point of view, was dismissed by the statement that it was an incurable disease. When he (Mr. Cheyne) worked up the subject for the Harveian Lectures six years ago he could not find any statistics, with the exception of some cases by Dr. Herringham, which at all indicated curability of the disease. The surgeon is, of course, entirely dependent on the physician for statistics of the prognosis of this condition in the absence of surgical treatment; and when surgeons were told by physicians that tuberculosis was an incurable disease in their hands they could only assume that their statement was correct, and draw any conclusions as to what followed surgical measures on the foundation of that statement.

The surgical treatment of peritoneal tuberculosis arose as the result of a mistaken diagnosis, for surgeons, like physicians, sometimes make such mistakes. When one surgeon after another had made such a mistake which had led them to open the peritoneum, and when it was found that patient after patient, the subject of this disease, tuberculous peritonitis, said by the physicians to be incurable, recovered, it certainly implied to their minds that the recovery was due

to their intervention; and hence it came about that the surgical treatment of peritoneal tuberculosis was established. The whole superstructure of the treatment by laparotomy, however, rests entirely on the physicians' statement that the disease was one that was not amenable to medical remedies. This evening, however, they had heard an acknowledgment by the physicians that they have not been right in this view, and that peritoneal tuberculosis is a disease very amenable to curative treatment without any operation. Under those circumstances, he (Mr. Cheyne) did not see that he could discuss the advantages or disadvantages of operation at the present moment. He must wait till we know whether other physicians confirmed this very positive opinion which had been enunciated by Drs. Sutherland and Guthrie; for if it is correct, naturally the operation of laparotomy, as an operation in the general treatment of tuberculous peritonitis, will fall to the ground. As Mr. Cheyne had already said, surgeons have been performing laparotomies all these years and founding statistics on them because the physicians not only told them in their writings that they could not cure the disease, but actually brought the cases to the surgeons to operate on. He (Mr. Cheyne) had never had to do with a case of peritoneal tuberculosis in the first instance; the cases which he had had were always brought to him by a physician after the physician had been trying to cure the case for months, and had at length come to the conclusion that his attempt was a failure. It had always been a mystery to him why such cases should recover after opening the abdomen, and he had tried a variety of theories to explain it. As a matter of fact, in all cases in which he had opened the abdomen the condition of matters looked so very bad that he had always given the friends a rather hopeless prognosis, and yet in a considerable number the patients had recovered. It struck Mr. Cheyne as a very curious thing that physicians had not discovered this curability of peritoneal tuberculosis before now, and that when the physician got tired of watching such cases and came to the surgeon the surgical treatment

coincided with the commencement of the recovery. In conversation Dr. Sutherland had indicated with regard to peritoneal tuberculosis that it ran a definite course, getting worse for a time, and then a period came when recovery began. He (Mr. Cheyne) did not know whether this was anything more than a theory; all that he could say was that it was very remarkable that this change in the course of the disease coincided with the period when the physician had got tired of watching these cases and brought them to the surgeon.

When he heard of the remarkable change of front on the part of Dr. Sutherland and Dr. Guthrie, he thought that some new remedy had been introduced, that they had discovered some fresh drug which cured peritoneal tuberculosis, but he found that that was not the case. What had happened was that they had learned to diagnose the disease. It appeared that physicians had been making mistakes in the diagnosis all these years, and that as the result of the efforts of the surgeon they had discovered that many cases of chronic inflammation of the peritoneum which they had previously looked on as of simple origin were in reality tuberculous. Hence, in former years a great many cases of tuberculous peritonitis were excluded from the diagnosis of tuberculosis, and only the worst and most hopeless cases were so recognised. Mr. Cheyne was not disinclined to agree with the physicians as to this mistake that they had been making in the diagnosis, and, as he had already said, he considered that the situation was now a new one altogether, and that while he would even now not refuse to perform a laparotomy in any case of tuberculous peritonitis brought to him by a physician where the long continuance of medical treatment had failed to ameliorate matters, he did not feel that he could strongly advocate the use of surgery in this condition till further statistics indicating the real prognosis had been collected.

When, however, Dr. Guthrie says that surgical treatment is of no value, Mr. Cheyne was inclined to disagree with that view. Whether it was a necessity for the patient's

recovery was a point that he was quite prepared to hold open to question till the physicians had settled the prognosis of the disease, but that improvement undoubtedly followed surgical treatment he was perfectly convinced. His experience seemed to point to the fact that some conditions in the peritoneal cavity were altered by the operation, with the result that improvement followed, and he could not look on the frequent improvement which did follow even in bad cases as always a coincidence. As a matter of fact, physicians were so accustomed nowadays to brilliant results when their cases got into the hands of surgeons that they expected the same thing to happen in every instance. He (Mr. Cheyne) must confess that he had never seen any sudden improvement follow laparotomy in peritoneal tuberculosis, and it is probably the absence of this sudden improvement that had led Dr. Guthrie to his very drastic conclusion. In Mr. Cheyne's experience the beneficial change had been a gradual one.

While, however, he was prepared to suspend his judgment as regards the general use of laparotomy as a method of treatment of tuberculous peritonitis, there were of course many cases of this disease where surgery was necessary to save the patient's life, more especially cases of obstruction, and where even Dr. Guthrie would hardly dispute that the surgeon was of some little value.

He was much interested in Dr. Sutherland's statistics, and it occurred to him as Dr. Sutherland was talking that something else might be proved from that gentleman's statistics than what he had deduced. Dr. Sutherland said very accurately that 41 cases were treated and 29 recovered. He then went on to contrast medical and surgical treatment, and he said that so many cases were treated medically with 81·3 per cent. of recoveries, and so many surgically with 50 per cent. recoveries. But, Dr. Sutherland forgot to mention that the cases which he brought to the poor surgeon were the bad cases; at any rate, those which had been brought to Mr. Cheyne by Drs. Sutherland and Guthrie were all of them very bad cases,

and he thought that he could safely conclude that they kept the best cases for themselves. He therefore submitted that this contrast between the cases treated medically and the cases treated surgically was not a fair one. What really was the fact from these statistics was, that of cases which went on so well that surgical interference did not seem to be desirable 81 per cent. recovered; while of those which were doing badly and were submitted to the surgeon 50 per cent. recovered, and he considered that that was a very admirable result of surgical treatment.

He would like to make another alteration in Dr. Sutherland's statistics. That gentleman said that 19 cases of those which recovered were under observation for more than a year, but of these 19 cases 7 were surgical cases which recovered entirely. Subtracting 7 from 19 leaves 12 out of 27 cases treated medically which recovered and were under observation for more than a year, so that it works out that of the cases under observation for more than a year 44 per cent. had recovered under medical treatment, while 50 per cent. had recovered under surgical treatment!

(*Read at the Special Meeting for the discussion of Tuberculous Peritonitis, December the 12th*, 1902.)

Discussion.

Dr. Robert Hutchison wished to bring forward one point in connection with the discussion, namely, as to whether the meeting was not running the risk of confusing two conditions which were essentially different: in the first place, tuberculous peritonitis occurring *per se* where the disease was primarily in the abdominal organs and remained there; and in the second place, tuberculous peritonitis occurring simply as a phase of generalised tuberculosis. He thought the value of the many interesting papers to which the members had listened that afternoon had been somewhat detracted from by a failure to recognise that distinction. In no part of the discussion was it more important to recognise such a distinction than when dealing with prognosis, because he thought all would admit that general tuberculosis was almost always fatal. Probably all would also agree that tuberculosis, as long as it remained limited to the peritoneal cavity and abdominal organs, offered unusual chances

of complete recovery. He admitted with Dr. Sutherland that one ought to speak cautiously about recovery in connection with tuberculous peritonitis, because it was possible for that condition to become generalised later, after the disease had been quiescent for a time. He wished to ask if any one present had any explanation to suggest as to why in abdominal tuberculosis the prognosis should be so much better than when the disease was elsewhere, such as in the lungs. He had heard, or read somewhere, that evidence had lately been produced in Germany to show that abdominal tuberculosis in children was really bovine tuberculosis, and that it was a different thing from the tubercle starting in most other places. It was suggested that that was why the prognosis was so much better than when the disease attacked the lungs.

With regard to the ascitic form of the disease, he asked whether there might not be a form of simple inflammatory effusion into the peritoneum which could not be distinguished clinically from tuberculosis. At one of the German Congresses the possible existence of such a form was admitted, and he would like to hear whether any one present had any proof to offer that there was such an inflammatory effusion which might be mistaken for tuberculous disease, and so account for the more favourable prognosis which tuberculous peritonitis seemed to offer.

Dr. Christopher Heywood (Irlams-o'-th'-Height, Salford) said he supposed he was right in stating that no drug had yet been found which had any decided effect upon tubercle bacilli in the body. Our object must therefore be to so improve the nutrition of the tissues that the cells could perform again the action which they normally did in health, and become capable of resisting and destroying the bacilli.

Even though cases of tuberculous peritonitis at times recovered without any treatment at all, yet physicians should do their best to help the tissues to overcome the micro-organisms. He wished to bear his testimony to the value of arsenic, a drug which acted powerfully on trophic nerves. The form of arsenic which he had used for some years, and which he found very valuable, was the combination of bromide of gold and bromide of arsenic; this rarely produced vomiting or diarrhœa, and acted as a strong tonic. The gold appeared to assist the arsenic in improving the nutrition of the cells and so increasing the resisting power of the tissues.

Mr. F. C. Abbott said there was no further occasion, after Mr. Watson Cheyne's remarks, to defend the surgical against the medical treatment of the condition. Probably all knew of cases in which the physician had himself tried surgery in so far as tapping the abdomen went, and in which laparotomy was performed afterwards. Of course, both Dr. Guthrie and Mr.

Watson Cheyne agreed that surgery was required in the more grave cases. He would have liked Mr. Cheyne's opinion as to what should be done in cases where there was obviously ascitic fluid over the front of the abdomen, but behind that a matting of intestines with abscesses and pus-laden loculi. On finding such a case should one go further, or be content with evacuating the fluid? By proceeding further one risked that serious trouble, a tuberculous fæcal fistula. His experience was that it was better to leave such a case alone.

Mr. R. H. Anglin Whitelocke (Oxford) had operated on only one case of tuberculous uterine appendages in a child. With regard to those cases in which there was matting of the intestines, he had followed Mr. Abbott's practice of leaving the adhesions alone, and had often been astonished to see such cases recover, even where there had been adherent omentum, and without their subsequently suffering from intestinal obstruction. In such cases he simply put in a gauze drain for twenty-four hours, and sealed up the abdomen as soon as possible afterwards. It was not wise to leave the drain in longer, on account of the risk of a tuberculous sinus. One case was handed over to him by a physician in which the whole peritoneal cavity was filled with serum, and it seemed a hopeless one, yet it ultimately recovered. Such cases surely led one to believe that surgical measures were necessary for the disease.

26. A CASE OF SYPHILITIC ENDARTERITIS AND NEPHRITIS IN AN INFANT.

By G. A. SUTHERLAND, M.D.,

AND

J. W. THOMSON WALKER, C.M.

A. C—, a female sixteen months old, was admitted into Paddington-Green Children's Hospital on September the 20th, 1901. The parents were Italians, and the father, who was said to have had syphilis, had died of heart disease. There had been one other child, who was still-born. The patient had appeared to be healthy up to the age of six months, when the right eye was noticed to be inflamed; the left soon became similarly affected, and she began to lose flesh. During the two months before

admission she had wasted rapidly, and had taken in the way of nourishment only a little beef tea.

On admission, she was distinctly emaciated, weighed 12 lbs., and presented evidence of congenital syphilis in the wizened face, the muddy complexion, and the sores around the anus and on other parts of the skin. She lay in a semi-comatose condition, taking no notice of anything, without voluntary movement, crying slightly only when disturbed, and never smiling. Owing to severe double ulcerative keratitis, with perforation of the cornea in the left eye, she was unable to see, and it was impossible to examine the discs. There were slight but definite evidences of rickets in the long bones. The right forearm was bent about the middle, the result evidently of an old green-stick fracture. The right arm lay motionless, apparently paralysed, and did not respond to any change of position. In marked contrast to this were curious movements of superpronation in the left arm, suggesting athetosis. Sensation was dulled generally, and the plantar reflexes were active. The spleen was enlarged and extended about an inch below the ribs. The cardiac action was weak, but there was no evidence of organic disease. The lungs and liver and temperature were normal. Mercurial treatment was adopted.

During the seventeen days that she lived in the hospital there were few changes to be noted. At times she passed a considerable amount of blood and mucus *per anum*, without other evidence of intestinal disturbance. The right arm became rigid, and the same condition extended to the lower extremities, the toes and feet becoming fixed and pointed. There was also definite retraction of the head. On one occasion there was retention of urine, and urine drawn off by the catheter was pale in colour, and contained a small amount of albumen. On another occasion she had an attack of hyperpyrexia, the temperature rising to 106·4° F., with signs of extreme collapse. There was no improvement under the mercurial treatment, and she sank gradually with increasing coma and rigidity, the latter condition being very marked before death.

Post-mortem report.—The body was emaciated. On removing the dura mater a quantity of clear, straw-coloured fluid was present in the subarachnoid space, being most abundant over the left frontal lobe. The left frontal lobe was small and shrunken, the convolutions in front of the fissure of Rolando being crowded together and greatly diminished in size, and the substance of the lobe soft and diffluent. The lateral ventricles contained a slight excess of clear fluid. The cut end of the left

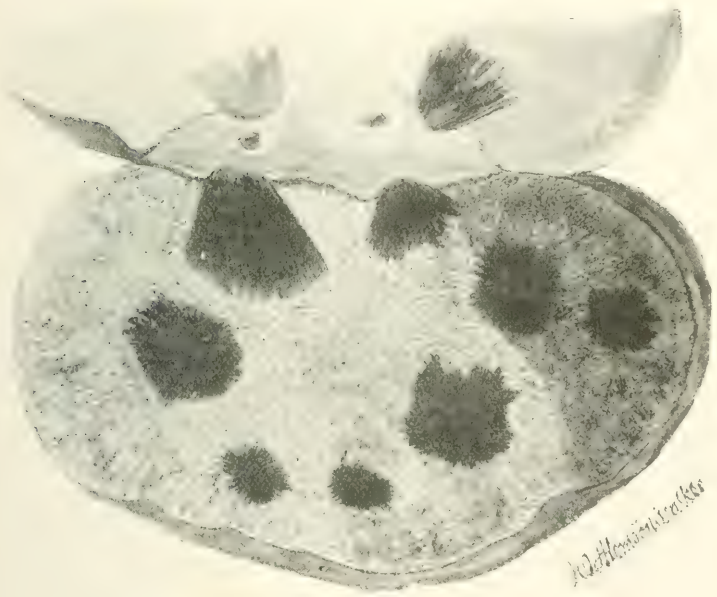
FIG. 17.

internal carotid artery projected rigidly from the upturned base of the brain, its lumen being open, and the wall thick and white. There was also thickening and rigidity of the wall of the left middle and anterior cerebral arteries, and a small segment of the basilar artery showed a similar change. On microscopic examination of the left middle cerebral artery the pathological changes seen in Fig. 17 were observed. In the adventitia, a wedge-shaped area with its base outwards, comprising about one eighth of the circumference, was infiltrated with small round cells, and the vasa vasorum were here dilated and increased in number. This change was present on the corresponding

part of the tunica media, and completely obscured the muscle-fibres.

The fibres of the elastic lamina, corresponding to this infiltrated area in the outer and middle coats, were separated from each other by small round cells, the elastic layer being broader but less compact. At another part of the artery, a well-marked strand of elastic tissue, appa-

Fig. 18.



rently of new formation, penetrated into the thickened tunica intima.

The changes in the intima were more marked than those in the other coats, and were not confined to one segment of the circumference. The endothelium showed abundant proliferation, but the greater part of the intima had the appearance of granulation tissue, the capillaries being derived from the infiltrated area of the outer coat.

The right kidney was much enlarged and deeply fissured. The capsule stripped easily, and exposed a surface stippled with yellow dots and a few pea-sized yellow areas. On section (see Fig. 18), only the upper pole of the kidney, about one fifth of the cut surface, was normal in appearance. The rest of the cortex had a brilliant pinkish-yellow colour, with here and there small wedge-shaped or irregular areas of apparently normal kidney tissue. The limit of the change in the cortex was

Fig. 19

sharply defined from the normal kidney tissue. The pyramids were of a dark reddish-brown colour, contrasting strongly with the bright yellow cortex.

The renal pelvis and arteries were normal.

On microscopic examination, the interstitial tissue of the cortex was densely infiltrated with round cells (Fig. 19). Here and there the infiltration was so dense that the tubules and glomeruli were obscured. In other areas, for the most part subcapsular, the infiltration was

absent. Fine capillary vessels were found in the infiltrated areas, and here and there their contents had escaped into the surrounding tissues. The adventitia of the larger vessels was infiltrated by closely packed small round cells, but the media and intima were unchanged.

The kidney tubules were widely separated by the infiltrated interstitial tissue. The lining epithelium was somewhat granular in appearance, and in many tubules the lumen was filled with casts of homogeneous material or masses of round cells. In the patches of dense infiltration the epithelium was shed and blocked the lumen of the tubule. A few of the glomeruli showed a slight increase in their nuclei, but the majority displayed no change. In the medulla round-cell infiltration was present, but only to a slight degree. In this part of the kidney there was engorgement of the blood-vessels, and the straight tubules contained casts of round cells.

The left kidney was slightly increased in size. The changes in its substance were similar in character to those in the right kidney but much less marked in degree.

Since the completion of this paper another case of congenital syphilis has been examined in the post-mortem room at the Paddington-Green Children's Hospital, and disease of both kidneys was present. This patient was a female infant, eight months old. When first seen she had a small lump over the right frontal region. She was ordered half-grain doses of grey powder twice daily. A week later she was found to be suffering from an acute septic sore throat, general purpura, and hyperpyrexia. She died within twenty-four hours of admission.

There was nothing in her family or personal history pointing to syphilis, but her appearance at the first examination was considered diagnostic of that affection.

Post-mortem examination.—The ossification of the cranial bones was remarkably incomplete, the various bones of the cranium being widely separated. A space the breadth of two fingers separated the squamous portion of the temporal bone from the left parietal, the gap being

bridged by membrane. There was a large collection of clear straw-coloured fluid in the cisternæ basalis and pontis, and the lateral ventricles were distended with similar fluid. A circumscribed patch of yellow lymph about the size of half a crown was present on the under surface of the right temporal lobe. The arteries of the circle of Willis and brain showed no abnormal changes to the naked eye. There was sloughing pharyngitis and tonsillitis.

Left kidney.—This was slightly enlarged; its capsule stripped easily, exposing a healthy surface. On section, the cortex was of normal breadth. The cut surface as a whole was pale in colour, with here and there pale yellowish patches in the cortex. These patches were most marked at an area midway between the upper and lower poles, and the convex border of the kidney was more prominent over this part. The patches gave the impression of an infiltration of the cortex, and were unlike the colour seen in fatty changes. At the bases of the pyramids there were well-marked, dark, hæmorrhagic spots. The pyramids were normal.

Right kidney. This was not enlarged; the capsule stripped readily. The cut surface was pale throughout, but showed none of the patches of infiltration seen in the left kidney. There were, however, several spots of ecchymosis similar in position to those in the other organ.

The microscopic examination of the kidneys demonstrated changes similar to those found in the previous case. There was the same diffuse infiltration of the cortex with round cells, the change being, however, more uniform without the patches of dense infiltration seen in the first case. The glomeruli and tubules were normal, but were widely separated by the infiltration. The epithelium of the tubules was unchanged. The vessels of the cortex were engorged with blood. Infiltration was present also in the interstitial tissue of the pyramids, but was much less marked, and towards the apex of the pyramid was entirely absent. There were none of the changes found in chronic nephritis.

In the first case, there are two pathological conditions, each of them of interest when considered alone, and both rendered doubly interesting by their combination in the same case.

Let us take first the disease of the cerebral arteries. The condition is one of arteritis obliterans of the cerebral arteries alone, occurring in a syphilitic child, and is therefore an example of Heubner's syphilitic endarteritis. Microscopically, there is nothing to distinguish this condition from a similar obliterative process which takes place in chronic inflamed areas, in arteries after ligature, and in the neighbourhood of tumours, and the diagnosis, therefore, rests upon the localisation of the arteritis to the cerebral arteries, the history of syphilis, and the absence of any of the other causes just mentioned.

The process was considered by Heubner* to be primarily a proliferation of the tunica intima, and he believed that the changes found in the muscular tunic and the adventitia were secondary, and were by no means an integral part of the process. Friedländer† and Baumgarten,‡ on the other hand, refer to the adventitia as the starting-point of the process. We are inclined to agree with the latter authorities in laying greater stress on the changes in the vasa vasorum of the adventitia than the proliferation of the endothelium.

It is certainly possible to cut sections in which no change is apparent in the middle and outer coats, but it will be found that in other sections in the immediate neighbourhood there is well-marked infiltration around, and increase in number of the vasa vasorum.

The process appears to consist in the production of granulation tissue in the vessel wall which begins in the outer coat, but only reaches its greatest development in the intima. The vessels of this granulation tissue are derived from the vasa vasorum, a very easily observed fact,

* 'Die luetische Erkrankung der Hirnarterien,' Leipzig, 1874.

† 'Centralbl. f. d. med. Wissensch.,' 1876, No. 4.

‡ 'Virchow's Archiv,' lxxiii, S. 90, 1878.

and it is not unreasonable to suppose that the inflammatory phenomena commence in the vasa vasorum themselves. Changes in the elastic lamina are very constant, and it has been said that the development of a secondary layer of elastic tissue, such as our sections show, is very characteristic of the syphilitic form of the obliterative arteritis. We are not, however, convinced that this phenomenon has much diagnostic value.

Syphilitic arteritis is an uncommon complication of congenital syphilis.

Heubner's original cases were all adults, and so were the cases on which the observations of nearly all the subsequent writers were based. Only a few isolated cases of arteritis syphilitica in congenital syphilis are found in the literature, such as those of Barlow* and Chiari.†

The occurrence of this process in the early months of congenital syphilis raises a point of some interest. Syphilitic arteritis is usually looked upon as a tertiary manifestation, and there is no doubt that the condition, with or without the presence of gummata in the immediate neighbourhood of the affected arteries, may coincide with tertiary lesions and chronologically come into this period. There is, however, undoubted proof that the condition may occur during the secondary period, accompanied by symptoms of secondary syphilis, and we have seen sufficient evidence, post-mortem and clinical, to feel convinced that it has, perhaps, more claim to be considered a secondary syphilitic process than a tertiary. Such cases as the one under consideration certainly give support to this view.

When we turn to the renal changes we are on ground common to the two cases. In both cases the process consists of a diffuse infiltration of the interstitial tissue of the cortex, and in both cases there is undoubted proof of congenital syphilis. Dr. Stoerk, of Vienna,‡ has recently described the effect of syphilis upon the kidney during

* 'Trans. Path. Soc. Lond.,' 1877, xxviii, p. 287.

† 'Wien. med. Wochenschr.,' 1881.

‡ 'Wien. klin. Wochenschr.,' 1901, No. 41.

fœtal life. An arrest of development takes place, or at least the growth is retarded, so that at birth the outer layer of cortex contains imperfectly developed tubules and glomeruli. Such, then, is the condition which may truly be called the congenital syphilitic kidney.

In the two cases we present the kidneys are fully developed, and the condition in no way corresponds to that described by Stoerk. They are cases of interstitial nephritis occurring in the subjects of congenital syphilis, and what we have to consider is how far the syphilitic poison is responsible for the production of the condition.

It is recognised that the ætiology of nephritis in childhood is in many cases extremely uncertain.

Dr. Leonard Guthrie,* in 1897, when discussing eight cases of chronic interstitial nephritis in childhood, insisted upon this uncertainty. More recently Dr. Guthrie† reported a case of chronic interstitial nephritis due to congenital syphilis, and he says, "On the whole, although evidence is far from being conclusive, I am inclined to think that chronic interstitial nephritis in young children is due to hereditary syphilis."

Massalongo has also fully described a case of chronic interstitial nephritis in a syphilitic child.

On the other hand, cases of acute interstitial nephritis in congenital syphilis are described by Coupland § and Stroebe. ||

If we look at the post-mortem records of cases of congenital syphilis we get some idea of the frequency with which renal disease is found in this connection. Thus we find that Speiss¶ has collected 34 cases, 10 of which had renal disease.

* 'Lancet,' February 27th and March 13th, 1897.

† 'Reports of The Society for the Study of Disease in Children,' vol. i, 1901, p. 69.

‡ Internat. Congr. Rome, 1894, 'Med. Rec.,' April 28th, 1894.

§ 'Trans. Path. Soc. Lond.,' 1876, xxvii, p. 303.

|| 'Centralbl. f. allg. Path.,' vol. ii, 1891, p. 1009.

¶ "U. die verschiedenen nierenaffektionen bei Syphilis constitutionalis," 'Inaug. Diss. Berl.,' 1877, quoted by Stroebe.

The association of renal disease and congenital syphilis is therefore not very uncommon, and a certain number of cases are on record in which interstitial nephritis, either acute or chronic, has occurred in these subjects without any very evident exciting cause.

The importance of syphilis as the causative factor in these cases cannot, we think, be disregarded; we would go further, and state our belief that the sole cause in many cases of interstitial nephritis in children is the syphilitic poison.

Nor does the importance of this question of etiology cease with these cases occurring in childhood.

The early changes in the granular kidney of adults are still speculative and the cause obscure. We are in accord with Dr. Guthrie when he suggests that hereditary and acquired syphilis may account for some of these cases also.

It is possible, and we believe probable, that many cases such as those we have described have been passed over in the post-mortem room. For although in one of the kidneys of the first case the changes were very striking to the naked eye, yet in the other kidney they were but slight, while in the second case only at one part of the left kidney was there any change likely to arrest the attention, and this did not by any means lead one to expect the extensive infiltration revealed by the microscope. Further, we read that in two of the cases described (those of Coupland and Stroebe) the kidneys showed no changes to the naked eye.

We would therefore submit that syphilis plays a more important and frequent *rôle* in the etiology of nephritis in childhood, and perhaps also of adult life, than has hitherto been recognised. (*Read January the 19th*, 1903.)

Discussion.

The Chairman (**Dr. George E. Shuttleworth**) (Richmond), in thanking the authors for their valuable contribution, suggested that members might with advantage compare their experiences

with regard to interstitial nephritis occurring in subjects in whom there was reason to suspect hereditary syphilis.

Dr. George Carpenter, who said that for some years he had been collecting clinical material, remarked as to the rarity of syphilitic nephritis in infants, in his experience, and related his own observations on that condition. The history of one case under his care in November and December, 1889, was as follows:* It was that of a weakly-looking female infant, aged five weeks. A rash appeared when she was two weeks old. When he saw her there was a muco-purulent discharge from the nostrils, and she was desquamating about the palms, the soles, the abdomen, and the ears, and to a less extent about the trunk and limbs. There were shallow ulcers on the lips, a yellow crust at the root of the nose, and yellow scabs on the ears. On the buttocks were some papules with crater-like centres. Her organs were normal, and the eyes and buccal cavity were free from disease. The urine was not examined. She was ordered Hydrargyrum cum Cretâ in grain doses thrice daily. A week from the time of her first attendance the back of the left hand was found to be puffy. The following week there was a deep ulcer with a yellow unhealthy-looking base in the natal fissure. Exactly three weeks from the time he first saw her both eyelids became œdematous, they were white and waxy-looking, and there was then pronounced dropsy of the feet, the legs, the hands, and the forearms. He drew off her urine by catheter, and found albumen in it, and examined by the microscope there were hyaline, epithelial, and a few blood-casts, but no free red blood-cells were detected. Whilst under observation she was treated with mercury, notwithstanding the kidney trouble, and without obvious untoward results. She died, and at the post-mortem examination both lower lobes of the lungs were pneumonic. The kidneys were lobulated—a not uncommon appearance in infancy, and in regard to their naked-eye semblance, they *seemed quite natural*, with the exception that in some places there was injection of the interglobular vessels. The microscopical lesions, which were advanced, were patchy in distribution, and were tubular and interstitial, a mixture of catarrhal and fibro-cellular changes, the latter predominating. In some instances the glomeruli were large and over-nucleated, and their capsules thickened. He drew attention to the very early age of his infant patient, and to the probability of the condition having started *in utero*. He had seen other clinical examples. One was that of a female infant aged six months, who had an extensive and typical syphilide. There was extreme general dropsy when he first saw her, and slight swelling of the extremities had been noticed for six weeks. He passed a

* 'Syphilis of Children,' by George Carpenter, M.D., *vide* pp. 56, 57, A.D. 1901.

catheter, but the bladder was unfortunately empty. She did not attend the hospital again, and he lost sight of her, but from her serious condition he had no doubt she succumbed to the disease.

Dr. Leonard Guthrie said he had felt special interest in the case brought forward by Dr. Sutherland and Mr. Walker, because the views expressed by the authors bore out, to some extent, the ideas he brought before the Society some two years ago. When describing a case of chronic interstitial nephritis in a girl aged seven years, he suggested, though he had very little to go upon, that the condition might be due to syphilis. Otherwise it was difficult of explanation. In children it was almost impossible to attribute chronic interstitial nephritis to the ordinary causes, such as gout or alcohol, nor yet to post-scarlatinal or parenchymatous nephritis. Acute interstitial nephritis in children might be fatal, but in less severe cases the subjects of it might live on to a considerable age, and ultimately die of granular kidney, due to hereditary or perhaps acquired syphilis. He thought the argument was supported by statistics from lunatic asylums. It had been stated that at Colney Hatch Asylum 70 per cent. of the cases which died had granular kidney. By far the largest proportion of deaths in lunatic asylums were due to general paralysis of the insane; and it was now well known that general paralysis of the insane was a syphilitic affection, and it was probable that the granular kidney which affected those patients was also syphilitic.

Dr. G. A. Sutherland said that in Dr. Carpenter's case that observer had the advantage of making a diagnosis during life. In Dr. Carpenter's case there was œdema, but not in theirs. The former seemed a mixed case, for the glomeruli were considerably changed, whereas in Mr. Walker's and his case no such change was discernible. He was glad to hear Dr. Guthrie's remarks, for his writings had put Mr. Walker and himself upon the investigations they had been pursuing on the subject. In the first case reported in the present paper the child was admitted into hospital in an unconscious condition, and remained so to the end. The opinion was hazarded that it might be endarteritis or cerebral thrombosis; but that was more or less of a guess, based upon the evident cerebral condition and the rigidity of the limbs. The child was too far gone for treatment to be effective, though it might be beneficial in a case which was recognised earlier. He knew Dr. Guthrie rather hesitated to employ mercury in such cases, because he was afraid of its effect upon the kidney; but in a case of pure interstitial nephritis he (Dr. Sutherland) did not think mercury would do much harm.

Mr. Thomson Walker said in Dr. Carpenter's case there were no changes evident to the naked eye, and he thought quite a number of such instances escaped detection because there was nothing obtrusive to arrest attention.

27. TRAUMATIC CEPHALHYDROCELE.

By J. W. THOMSON WALKER, C.M.

This specimen illustrates a condition entirely confined to childhood.

The case was a boy of 13 years who received a blow on the head producing a scalp wound. The wound rapidly healed, but some months later a pulsating swelling appeared at the seat of injury. This had all the characters of a fluid swelling communicating with the interior of the cranium. After some years the tumour diminished and finally disappeared. The boy died of scarlet fever.

A scar about one inch in length was found in the scalp. The calvarium shown displayed the following appearances. In the line of the coronal suture to the left of the middle line is an irregular gap about $1\frac{1}{2}$ inches long, and varying in breadth from $\frac{1}{16}$th to $\frac{6}{16}$ths of an inch. The inner half of the gap follows the coronal suture, the outer half passing outwards and backwards into the left parietal bone. From the outer limit of this the remains of a repaired fracture can be traced for another $1\frac{1}{2}$ inches, where it again joins the coronal suture. This part of the fracture shows almost complete repair on the outer surface, only two small gaps being left, but on the inner surface the attempt at repair is very slight. The edges of the large gap are smooth and rounded, the anterior edge being bevelled at the expense of the inner table, while the posterior is bevelled at the expense of the outer table. On the inner surface of the frontal bone there are numerous pits and depressions of varying size, apparently due to pressure. Beneath the gap in the cranium the dura mater was deficient, and there was evidence of old-standing injury to the cerebral substance.

The condition of traumatic hydrocele is uncommon, and when present is usually cured by pressure, so that an opportunity of examining the bones does not often occur.

It has been suggested by Koenig that should pressure or aspiration fail to relieve the condition the bone should be exposed, and the pericranium stripped up and stitched over the gap. This has apparently not yet been attempted.

(*Read January the* 16*th*, 1903.)

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28. PUNCTURED FRACTURE OF THE SKULL.

By J. W. THOMSON WALKER, C.M.

The patient, E. B., a male, aged $7\frac{10}{12}$ths years, was admitted into the Paddington-Green Children's Hospital in a comatose condition, and died fifteen minutes after admission.

There was a history of long-standing otorrhœa following scarlet fever. Four days before admission he received a blow on the head with a broken whip with a projecting steel core, which caused a small scalp wound over the left parietal bone. There was free external hæmorrhage but no immediate cerebral symptoms. Two days later the child had a "fit" with twitching of the right side of the body, and the doctor who was called in made a diagnosis of meningitis due to middle ear disease, and sent the patient to the Golden-Square Throat Hospital. Dr. Lambert Lack sent him on at once to the Paddington-Green Children's Hospital.

At the post-mortem examination there was a small punctured wound in the scalp just below and in front of the left parietal eminence, and surrounding this a limited hæmatoma of the scalp. In the left parietal bone there was the small puncture seen in the specimen. A spicule of the inner table projected inwards. The track could be followed through the membranes, and on the surface of the parietal lobe there was a small puncture surrounded by ecchymoses. There was no sign of meningitis over the convexity of the hemispheres. A probe passed along a track in the brain

substance into the posterior horn of the lateral ventricle. For about an inch there was a well-marked track, but internal to this there was softening of the brain substance. There was early septic meningitis at the base, which passed backwards to the under surface of the cerebellum.

There was no trace of inflammation passing through the bony wall of the tympanum on either side. The left tympanic cavity contained some thick yellow pus but the right was normal. The principal interest attaches to the presence of middle ear disease on the same side complicating the diagnosis. (*Read January the 16th*, 1903.)

29. A CASE OF PARALYSIS OF THE CERVICAL SYMPATHETIC NERVE.

By SYDNEY STEPHENSON, C.M.

Henry H—, aged 5 months, was brought to my out-patient department at the North-Eastern Hospital for Children on January the 7th, 1903, because "his left eye had been getting smaller for a few days."

Upon examination, the child was found to have the signs characteristic of paralysis of the left cervical sympathetic nerve, viz. marked drooping of the left upper lid, some recession of the eyeball, a small pupil, and some reduction of the intra-ocular pressure. As regards the pupil, the inequality between the two eyes was much more marked in a dim light, inasmuch as under those conditions the right (unaffected) pupil dilated, while the left pupil (affected) did not respond to darkness. Hydrochloride of cocaine caused no appreciable alteration either in the amount of ptosis or in the size of the pupil. A puckered scab was present about one inch above the outer third of the left clavicle, immediately behind the posterior border of the sterno-mastoid muscle.

The history of the case was most instructive. It appeared

that on December the 20th, 1902, the child was brought to the hospital on account of a large and hard swelling in the left side of the neck. Fluctuation could not be definitely felt. A surgical colleague made an oblique incision over the tumour, and evacuated a deep abscess by Hilton's method. No bare bone could be felt, but as the abscess extended to the mucous membrane of the pharynx a diagnosis of retro-pharyngeal abscess was made. According to the mother's statement, nothing was noticed amiss with the baby's eye until the above operation had been performed. No amount of cross-examination failed to disturb her conviction. The child left the hospital, where he had been an in-patient, on December the 28th, 1902, with the wound practically healed. There was no rise of temperature during the time he stayed in the institution.

In view of the above facts, it is difficult to resist the conclusion that the operation and the paralysis were intimately connected. (*Read January the 16th*, 1903.)

Discussion.

Dr. Edmund Cautley asked whether there was any difference in the blood-vessels, or any difference in the function of sweating on that side.

Mr. Sydney Stephenson, in reply, said he would try to get the parents of the child to attend with it at the next meeting. He had observed no difference in the blood-vessels of the two sides of the face. The mother assured him the baby never sweated.

30. MICROCEPHALY WITH UNUSUAL MALDEVELOPMENT OF SKULL.

By LEONARD GUTHRIE, M.D.

Dr. Leonard Guthrie showed an infant, aged 5 months, who was subject to this deformity. The frontal bone was small, narrow, and retreating. The superciliary ridges,

zygomas, and temporal ridges were unduly prominent. The interfrontal suture was marked by a bony ridge. The parietal bones were small, and the left overlapped the right. No anterior fontanelle could be felt. The occipital bone showed a beak-like projection, and overlapped the two parietal bones. Thus the deformity of the skull seemed to be secondary to ill-development of the brain. The child's features were normal and did not suggest imbecility. No other deformity was present, but there was general spasticity of all the limbs. The arms were flexed and the fingers clenched, so that the nails had abraded the skin of the palms. The lower extremities were rigid and the reflexes exaggerated.

There was no history of a similar, or of any congenital deformity in other members of the family.

(*Exhibited January the 16th*, 1903.)

Discussion.

Mr. A. H. Tubby entered a protest against operating upon such children as Dr. Guthrie had shown. His own experience was that some years ago he operated upon four such cases. The first was rather a small baby of the usual type, and he died suddenly two days afterwards. He believed the second case was alive now. It was a very extensive operation, and he began the operation by taking out a linear strip of the left parietal bone, then crossed the middle line with difficulty, on account of the superior longitudinal sinus, and then came back to the front on the right side, making a horseshoe excision of bone. The child was no better for the operation, and is now in an idiot asylum. In the next case the bone was very thick, and he encountered an artery as large as an adult radial. This case was lost sight of. In the last case the operation seemed to have a better result, but the child had summer diarrhœa which proved fatal. It was an interesting pathological speculation as to whether the condition of the brain was primary, or whether the skull condition preceded it. His belief was that the two conditions were concurrent, because one frequently found in such cases the so-called "rabbit's intestine" brain and porencephalus. The condition of the skull was also very interesting. What was the cause of the rapid and abnormal ossification at the edges of the sutures? Where the suture should be there was simply a ridge of bone. So far as could be judged, craniectomy was quite useless to cure the condition.

Dr. Edmund Cautley said Dr. Guthrie's case was very unusual with regard to the shape of the skull, and in that microcephalic idiots did not always have spasticity of the limbs. He had had a similar case under care a considerable time. The head had considerably improved in shape. The child took its food even better than an ordinary child, and quickly gained weight. There was no spasticity of the limbs. Such cases could not well be classified by the shape of the skull alone. In some microcephalic idiots there was a defect in the development of the brain; but in others the condition was due to a true encephalitis, which might occur at birth or even afterwards. Considerable investigation of the subject was required, especially post mortem.

Dr. Robert Hutchison said he had recently seen two cases almost exactly like that shown by Dr. Guthrie, showing the peculiarity of sutures and considerable spasticity, though not so extensive as in the case shown. He had seen an unusually large number of such cases, which were mostly of Jewish parentage, and would be glad to hear if that was the experience of other members also. Many of his cases were drawn from the east end of London, but he had noticed the fact also in children elsewhere. Other forms of mental deficiency, however, were not common in Jewish children; for instance, Mongolianism was extremely uncommon among them.

Mr. George Pernet said that when he was assistant to Sir Victor Horsley at University College Hospital he had done craniectomy on five or six microcephalic children. One of two cases was of the Semitic type. In one instance the child lived about five and a half years after three operations, from which it derived no benefit. It died of some pulmonary affection. In the other the child had been lost sight of. In another case the type of defect was Mongolian, and the parents were English. One child had, in addition, cross-legged rigidity, and the upper limbs were flexed and fixed. The child was also blind. When craniectomy was performed he noticed the skull was very thick, and on accidentally pressing the brain substance the respiration was momentarily arrested. As soon as he removed his finger respiration was resumed.

Mr. Tubby said that when using Hoffman's forceps, after making the trephine opening, very slight movements caused temporary diminution of respiration, doubtless due to pressure on the cerebrum.

Mr. Pernet, continuing, said none of the children he had mentioned died from the craniectomy; there was no complication, and the wounds healed by first intention. In one case, that of a Mongolian type, there was an improvement. One of the children suffered from epileptic fits. This child is still living, and is now thirteen years old. Several operations were performed upon her

when she was between two and three years of age. Dr. J. H. E. Brock, under whose care the child was, had informed him that she still suffered from epileptic attacks, and that she had not been benefited the slightest by these operations. Those craniectomies were done when microcephaly was thought to be due to premature synostosis. He believed it more likely that the brain defect was the primary condition, and that the skull-cap adapted itself to the size of the brain.

The Chairman (**Dr. George E. Shuttleworth**) (Richmond) thought the anterior aspect of Dr. Guthrie's case conformed closely with the general features of microcephalic infants, and in that type the interfrontal suture was commonly to be made out. The good features and bright eyes generally coincided with a fairly good use of sight and smell, but not always of hearing. He thought there could be no question that the primary defect was in the formation of the brain. There was a want of development in the frontal regions, and arrest in the parieto-occipital region with imperfect development of the whole of the brain. He was necessarily interested in craniectomy at one time, as he was then medical superintendent of a large asylum for imbecile children, and was constantly being asked about it by parents who had read the tales in popular magazines giving instances of alleged remarkable cures by the operation. He had been to Paris to investigate the matter when M. Lannelongue was doing the operation, and although surgically the operations seemed successful, the psychological effect was practically *nil*. He, like Mr. Pernet, had indeed noticed slight improvement in some of the cases after operation, but he thought that was attributable to the careful nursing received over a long period. He saw in consultation some years ago a microcephalic child, aged seven years, in Cheshire, whose skull was about sixteen inches in circumference. There was also some paralysis, and the question was whether craniectomy should be recommended. From his experience he could not conscientiously recommend it, but was advised that the father was determined to have it done. He therefore said there were surgeons in London who believed the operation was beneficial, and advised in consultation with one of them. The operation was done, the amount of bone removed being the largest he had ever seen. First, an antero-posterior piece of bone three quarters of an inch wide was taken away from the forehead to the occiput at two sittings. At the third operation bone was removed at right angles over the left frontal convolution, in the hope that "the speech centre might be released." But no good came of it, for the child when seen one year and two years subsequent to the operation was worse rather than better. A similar unsatisfactory result was the outcome in three or four other cases he had had under observation. Nor could he understand of what advantage it would be

in Mongolianism, as there was no suspicion in that type of idiocy that there had been premature synostosis. He would have been glad if Dr. Guthrie could have thrown any light on the etiology of the case he brought forward. No doubt it was due to arrest in an early period of pregnancy, but he thought it had not been made out under what circumstances it occurred. He had only seen one microcephalic case whose parents were Jews, and that case came from the east end of London.

Dr. Leonard Guthrie, in reply, said he believed Virchow was of opinion that Mongolianism was due to premature synostosis of the basisphenoid, but Dr. Guthrie made a post-mortem examination in one case and found a distinct zone of cartilage at that site. He believed that in the cases under discussion the skull did not develop because the brain did not, and the two kept pace with one another. In some cases he thought there might be premature synostosis in one part of the skull but not in others. Where there was a more extensive synostosis of all the sutures there might be true microcephaly, in which case the microcephaly would be due to premature synostosis of all the sutures. He thought it possible for operation to do good in patients who were dull and lethargic by giving more room in the cranium, and one case had been benefited in that way. Operations in other cases in which premature synostosis was absent he thought were mostly employed for their moral effect, but moral effect could be produced without making holes in the skull. He was in accord with Mr. Tubby's opinion that operation in such cases was not justifiable. He thought it probable that the parents of the child shown were Jewish. Fits in association with microcephaly were very common, but he did not know that the present child had had them. He had not heard any cough by the child, but had seen the slight spasms of respiration which had been referred to; those were in keeping with the general spasticity.

31. DILATATION OF THE STOMACH IN A CHILD EIGHT YEARS OF AGE.

By CHARLES W. CHAPMAN, M.D.

S. W—, a girl aged 8 years, came under my care on December the 20th, 1901. The patient—an only child—was unusually bright and intelligent; she had been

accustomed to late hours, parties, unsuitable food, and sweets in abundance. She was overdone in her lessons by having special teachers at short intervals during the day, and in addition she was working hard at the violin. There was not much time for fresh air and exercise or sleep. She had been operated upon for adenoids and enlarged tonsils, and had had an enlarged cervical gland scraped.

The father was healthy, but the mother was liable to rheumatism and was under my care for mitral regurgitation.

History of present illness.—For the last two years the mother had noticed that the child's health was failing, the appetite at one time bad, at others ravenous; there was occasional vomiting, the bowels acted irregularly, and the motions were offensive. There was little or no pain after food. Finding the debility increasing, the common error of giving tonics in the form of chemical food was resorted to, the effect of which was to make the patient worse. Shortly before the child came under my care she had been on a visit to her grandmother, and while there had some dancing lessons. It was at one of these that the grandmother was alarmed at hearing a loud splashing sound as the child danced round the room. This was the symptom that made the parents seek medical advice.

On examination the child looked ill and careworn, there were dark rings round the eyes; the tongue was dirty, and the breath had a sour fermentative odour. The urine was stated to be often "sandy." On the patient being jogged a loud splashing sound was evoked, which could be heard across the room. There was little, if any, abnormal fulness at the epigastrium. The stomach resonance was up to the fourth rib in the left mid-axillary line; the lower border could not be defined, but it appeared to extend to within two fingers' breadth of the umbilicus. The lungs were normal and the heart healthy. No tumour could be made out in the abdomen, but there was marked splashing on palpation.

Taking into consideration the temperaments of the

patient and her mother, it was determined to postpone lavage and to try the effect of general measures. The number and duration of the lessons were strictly limited, and those which caused the greatest mental strain and excitement with consequent interference with sleep were stopped entirely. No food beyond a small glass of milk was to be taken between meals; fifteen minutes' quiet was enjoined before taking food, and thirty minutes' rest on the sofa with an entertaining book was ordered to be taken after meals. The diet was restricted to foods easy of digestion and least liable to fermentation. Thorough mastication was insisted upon, and no fluid allowed until near the end of the meal. If the child was thirsty, water, preferably hot, was allowed up to three quarters of an hour of mealtime. The medicinal treatment consisted of a mild mercurial purge twice or three times a week, and a mixture containing bismuth and sodium carbonate with nux vomica and a carminative shortly before each meal. No stimulant of any kind was allowed. Under this treatment there was slow but steady improvement all round, the splashing became less, the appetite and weight increased, and the complexion assumed a more healthy appearance. The child went for a prolonged visit to Scotland with judicious friends, from whom I had satisfactory reports from time to time. When seen early in November the patient was in perfect health.

Remarks.—This patient's condition was, in my opinion, due to improper feeding, and nervous exhaustion from overstudy and insufficient sleep. The interesting point is the manner in which the splashing was first noticed, and it suggests the probability that such cases, though with less obtrusive signs, may be more common than is generally supposed. (*Read January the 16th,* 1903.)

Discussion.

Dr. G. A. Sutherland thought the child was young for such a condition. It was that class of case which was associated with cyclic albuminuria. Children of nine to thirteen years of age had

a tendency to weakness of all the abdominal organs, and dilatation of the stomach was not uncommon. In most of the cases seen the dilatation was probably intermittent.

Dr. Edmund Cautley asked whether there was a history of attacks of abdominal pain, or anything pointing to congenital gastric spasm or pyloric spasm. Pyloric spasm had been referred to inco-ordination.

Dr. Charles W. Chapman, in reply, said no spasm was made out. He saw the child during the first two or three months, when a distinct splash was made out on each occasion, but it got less and less. The child was extraordinarily bright and intelligent but highly neurotic.

32. A CASE OF ACUTE PRIMARY THYROIDITIS IN AN INFANT.

By GEORGE CARPENTER, M.D.

Richard S—, a rickety and lachrymose infant with flushed cheeks, aged 14 months, was brought by his mother to the Evelina Hospital in December, 1900, for a swelling in his neck. This had been noticed a little over a week, and she thought it had appeared suddenly. Her attention was attracted to the child by his inability "to hold up his head," and by his desire "to lie undisturbed in his cot." There was nothing worthy of note about either his family or his personal history.

On examination I found a swelling in his neck the size of a walnut. It was situated over the trachea, and occupied the position of the thyroid gland. It was larger on the left side than on the right, and it moved with the trachea during deglutition. The skin over the tumour was freely movable and of healthy appearance. His temperature was 101·8° F. The pulse-rate was 152 to the minute, and the pulse was regular and of good volume. The throat was normal, and there was no evidence of either tracheal or laryngeal obstruction. The posterior nares contained adenoids. On January the 3rd, three days from his first

attendance, an abscess had formed which was treated surgically. On January the 17th the abscess cavity had greatly contracted, but there was still some slight swelling of the gland. He subsequently made a good recovery.

Acute thyroiditis is, in my experience, a very uncommon condition, and sufficiently so, I take it, to make it necessary to place examples of this complaint on record. Carl Stamm* has recorded a case of acute idiopathic thyroiditis in a girl of 11 years old. It was attended by fever, cough, hoarseness, and difficulty of breathing. The glandular swelling disappeared in eight days under treatment.

My case also was apparently a primary thyroiditis, for there was no history of it starting as a complication of measles, diphtheria, typhoid fever, parotitis, or other specific conditions, illustrations of which can be found scattered in medical literature.

(*Read January the 16th*, 1903.)

33. A CASE OF CONGENITAL CEREBELLAR ATAXY.

By G. A. SUTHERLAND, M.D.

This boy is now 4½ years old. He was born after an instrumental labour, in the course of which his mother suffered from convulsions. He seemed healthy at birth, and has enjoyed good health since, never having suffered from convulsions or any illness, except whooping-cough a year ago. He did not learn to walk until he was three years old, and his walking has always been very unsteady. His mother did not consider him specially backward in his development, but as he was the first child her experience had been limited. She has now another baby, four months old, and considers him much more forward than the present patient was at the same age. The family history is good,

* 'Pediatrics,' vol. ix, p. 508, A.D. 1900.

and there is no evidence of syphilis or other constitutional affection.

He first came under observation eighteen months ago, when the condition was as follows:—His body was well formed and well developed, and the expression of his face was intelligent. Walking was performed with difficulty, the arms being raised at the elbows to aid him in balancing himself, the feet being widely separated, and staggering and tumbling being frequent. When he started off to walk his head was deflected towards the right shoulder, and his face was rotated to the left side. He could not stand with his feet placed together. When sitting or standing there was general tremor of the body and limbs, which was much increased on walking. When placed on his back on the floor he was unable to raise himself to a sitting posture. His method of getting up was to turn (with difficulty) on to his face and push himself up with his arms and legs, an effort which frequently ended in a collapse on the floor. There was also inco-ordination of the upper extremities, specially marked in the finer movements of the fingers. There was no muscular wasting, or hypertrophy, or spasm, or tenderness.

His speech was slow, staccato, and blurred. The knee-jerks were present, active, but not exaggerated, and there was no ankle-clonus. The plantar reflex pointed to extension of the great toe, but was difficult to determine. There was no rectal or vesical trouble. No sensory changes could be made out. Vision seemed good, the pupils reacted normally, and there was no nystagmus. Ophthalmoscopic examination was negative.

During the past eighteen months there has been marked improvement in his standing and walking, and the tremors are less, but the above-mentioned symptoms although less in degree are still present.

Remarks.—In this case there would appear to be a congenital lesion characterised by tremors and ataxy. The occurrence of any purely muscular or spinal lesion at such an early age is unlikely. As regards an affection of the

cerebral hemispheres, it is possible that non-development or atrophy of certain cortical centres or subcortical areas would produce the symptoms which are present, but it would be of so selective a character that one cannot regard it as probable, and so far as I know the condition has not been observed. In other words, one would certainly find a more extensive area involved and other symptoms superadded. The resemblance to the signs produced by cerebellar disease are very striking. If we assume that there has been imperfect development of the middle lobe of the cerebellum or of the whole cerebellum, the functional disturbances would be readily explained. A point in connection with cerebellar lesions on which Dr. Hughlings Jackson lays stress is paresis of the muscles supporting the trunk. In this patient, although there is no lordosis, there is definite weakness of the muscles which flex and extend the trunk, and no corresponding weakness of the muscles of the extremities. Dr. Risien Russell in his experimental work found that there was a deviation of the head to one side in certain lesions of the cerebellum, and this was present in the patient now shown. Judging by the symptoms, I do not think we have to deal here with a progressive lesion. On the contrary, his walking power has much improved, and it seems probable either that development in the impaired centres is taking place, or that other centres are taking on the function of those which have been inactive since birth. The only treatment adopted has been to maintain the general health at the highest possible level.

(*Exhibited February the 20th*, 1903.)

Discussion.

Dr. Fletcher Beach thought, from the history of the difficult labour and the use of instruments, that some lesion of the brain might have been caused producing hæmorrhage. The lesions usually found in such cases were cerebral diplegia, which was absent in the present case. He did not remember any cases of difficult labour in which the cerebellum was supposed to be affected; in fact, he did not see how the cerebellum could be affected by the difficult labour and by the application of forceps.

whereas hæmorrhage and encephalitis might be so produced. He did not notice any mental deficiency about the child. He asked whether Dr. Sutherland thought the application of a constant current would be of any service.

Dr. James Taylor agreed that the lesion was probably cerebellar, and that it was likely to have dated from the time of birth. It was quite conceivable that there was a lesion of the cerebellum as a result of the labour. In a series published some years ago by Dr. Herbert Spencer, who made many post-mortems on still-born children, there were a number of cases of hæmorrhage in the cerebellum. If that occurred in still-born children he did not see why it should not occasionally occur in children born alive and continuing to live. Or it might be a case of congenital defect of the cerebellum similar to cases associated with the name of Marie, named cerebellar ataxy: but in the latter there were other symptoms associated with the ataxy, and the disease was not usually manifest until about twenty years of age. It was conceivable that there were cases in which a similar defect occurred in the cerebellum, but in which the symptoms of the case were seen much earlier than in Marie's disease. He recently had a case under observation in which the patient had ataxy and optic atrophy. The patient was a lad of 18, and the symptoms first showed themselves at ten years of age.

The Chairman (**Dr. Edmund Cautley**) asked Dr. Sutherland whether he had considered the possibility of there being any porencephaly in the cerebellum. Also, was the lesion unilateral, or one affecting the whole of the cerebellum?

Dr. G. A. Sutherland, in reply, thought the lesion might affect the middle lobe or the whole cerebellum. He questioned whether a lesion of the lateral lobe would produce symptoms so exactly bilateral. He had not thought of the possibility of it being porencephaly, a condition usually found post mortem, and but seldom diagnosed during life. He was very interested in Dr. Taylor's observations, and agreed that in the child shown the lesion was probably ante-natal, so that the same or similar symptoms to those which Marie described might be present. Both Dr. Beach and Dr. Taylor thought it possible, from the history given, that there might have been a lesion caused at the time of birth. Certainly instruments were used, but he did not know of any lesions of that precise nature having been recorded as associated with meningeal hæmorrhage. Labour was induced chiefly for the mother's sake: there was no history of cyanosis or other symptoms usually looked for in connection with meningeal hæmorrhage. He thought such hæmorrhage in infants was usually associated with general bleeding about the hemispheres, and was not localised to a small part of the brain. It was difficult to imagine hæmorrhage taking place only in the cerebellar region and not in other parts as well. He had not

tried the constant current. If one assumed a central lesion, would the constant current be likely to do much good? He would, however, give it a trial.

34. A CASE OF ACHONDROPLASIA.

By JAMES TAYLOR, M.D.

The case was characterised by the large size of the head and the shortness of the limbs. The trunk was comparatively normal, but there was a disproportion between the upper arm and the forearm, and the upper arm was very short. There was a similar disproportion between the thigh and the leg. The hands were short and broad, and the fingers could not be brought into close apposition, but spread out in the way described by the French observer Marie. The condition probably depended on a defect in ossification, and consequently there was a shortening of the limb. That also probably accounted for the bone which was formed being of a peculiarly thick and dense character. On lifting the child it was found to be enormously heavy compared with an ordinary child of the same height. There were seven children in the family, and the other six were quite normal. The mother did not know why the child should be so deformed, except that a child living next door had a very similar face, and she used to see it while she was pregnant!

(*Exhibited February the 20th*, 1903.)

Discussion.

Dr. Travers Smith (Kilburn) asked whether Dr. Taylor could explain the extraordinary prominence of the forehead and the great sinking-in of the bridge of the nose.

The Chairman (**Dr. Edmund Cautley**) said the condition was not common, and the cases usually came under observation for some other state of disease. He had read somewhere that achondroplasia was often associated with hydramnios, and perhaps Dr. Taylor could say whether that was present in his case.

The bones at the base of the skull were often affected in achondroplasia; consequently there was premature synostosis and deformity of the head for that reason. He believed it was suggested that the affection was due to some form of osteomyelitis. Such cases—at least in the old days—were often included under the heading of fœtal rickets. The abnormality was seen also in calves, sheep, and pigs.

Dr. James Taylor, in reply, said the cases were rare: that was the first he had had under his own care, though he had seen several others. He could not say why there was arching of the forehead and depression of the bridge of the nose, but it seemed clear that there was some perversion in the bone-forming function of the cartilage. Such cases had been known from time immemorial, and, as Marie had pointed out, some of them had been worshipped as gods, or at all events gods were made in the shape of achondroplasic dwarfs.

35. A CASE OF RAYNAUD'S DISEASE.

By ALEXANDER MORISON, M.D.

Since Raynaud described his cases of local asphyxia in 1862 and again in 1874, and collected cases from medical literature to illustrate his theories, the phenomena of the clinical condition which goes by his name have been closely studied, and some frequently concomitant conditions described, which appear to have escaped the notice of Raynaud and the early writers on the subject. Thus, nowhere does he mention the intermittent hæmoglobinuria at times associated with these cases, and Sir Thomas Barlow, in his excellent translation and commentary on Raynaud's papers,* refers to other clinical associations of the disease, which have been emphasised by later observers. The severity of these cases varies very much, being conveniently divisible into cases of (1) local syncope, as it has been termed; (2) of local asphyxia; and (3) of local gangrene. These three may all be concomitant and of little severity, or they may lead to destruction of parts and even to the death of the patient.

* Selected Monographs, New Sydenham Society's Publications, 1878.

The case I have to bring before you to-day is, at present, in the category of the milder cases, but it illustrates sufficiently some important considerations in connection with the etiology and character of the disease.

H. J—, 8 years of age, is the fourth and now eldest child in a family of eight. The first child was still-born, the second is stated to have died of a "laryngeal growth and pneumonia," and the third was prematurely born at the seventh month. Three children have been born since the patient, and are apparently well. The parents were twenty-three and twenty-one years of age respectively when they married and are stated to be healthy. The patient had "snuffles" as a baby but no skin eruption.

In November, 1899, he suddenly complained of pain in one leg and foot. Touching the part caused pain. The left foot and leg as far up as the knee, and the right foot were the parts which were at first affected, and he could not walk. His mother carried him to an omnibus, and thence to a doctor, who, it is stated, declined either to express an opinion as to the nature of the case, or to prescribe for the condition. The parts affected are stated then to have become cold and blue. The mother, deserted by the faculty, placed the patient in bed, and on allopathic grounds wrapped the cold blue parts in warm flannels. Next day it is stated that no complaint was made of the limbs, and the child went to school. Since then, every autumn, when the weather becomes cold, he complains of pain in the hands and feet, and loses power in them, and the parts become cold and blue. His ears also become cyanotic, as may be seen at present. Previous to the supervention of this condition the child had erysipelas when 8 months old, and "fits" when 15 months old. Two years ago he is also stated to have had whooping-cough, measles, varicella, and a "fit."

The patient was admitted into the Children's Hospital, Paddington Green, under my care on January the 28th of this year. He then had blueness of both hands and feet and of the ears, while on the left foot there were several

blood-stained bullæ, varying from the size of a sixpence to that of a split pea. After admission into the hospital the cyanosis of the limbs passed off under the influence of warmth, the bullæ dried, and some of the scabs have dropped off. The ears alone have retained their cyanotic hue along the upper and posterior part of the auricles. The organs generally are normal. There is no cardiac affection, congenital or acquired, nor is there any distress of breathing. The patient has not manifested any hæmoglobinuria, but a blood-count carefully made and repeated for confirmation by the house-physician (Dr. Carey) showed a hypercytosis of the coloured corpuscles by rather more than a million, the actual figures being 6,080,000. There was no increase of colourless corpuscles, but they were not carefully counted.

The case illustrates an etiological point which has frequently been observed in these cases, namely, a probably specific origin, the early parturient history of the mother being suspicious of that taint. It also exemplifies the symmetrical nature of the affection, and the influence of external cold in provoking it.

(*Exhibited February the 20th*, 1903.)

Discussion.

Dr. Fletcher Beach asked what treatment had been adopted in the case. He read of a case having been treated by the application of an Esmarch's bandage to different parts of the body. This, continued for three or four months, resulted in a cure. At first, on account of the pain, the application was limited to about two minutes at a time; but the relief was so great that the patient begged for its repetition, and eventually it could be applied for five-minute periods.

Mr. George Pernet said he had been interested in the statement about the formation of bullæ containing blood. Another point was that in sclerodermia, especially in the sclerodactylic form, the condition had been preceded by Raynaud's phenomena for a considerable time. It would be interesting to watch the case, perhaps for some years.

Dr. Theodore Fisher (Bristol) referred to an unusual type of case, one similar to Raynaud's disease, but with hæmorrhages in one hand as the result of a blow. The patient was a girl aged 9 years. Two years before he saw her she was said to have

received a severe blow across the knuckles at school with a ruler, and hæmorrhages developed soon afterwards. Two or three times a year she had pain and coldness of the hand, followed by hæmorrhages. The condition was still persisting, though it was about six years since he first saw her. It had been said that some central nerve disturbance of permanent character could be produced by strong peripheral stimulation. Possibly the blow on the hand in the case he had related might have caused some such central disturbance.

Dr. Leonard Guthrie believed it likely, from an examination of Dr. Morison's patient, that the condition was due to congenital syphilis: he had the typical physiognomy, though there appeared to be no interstitial keratitis or scarring about the mouth. The upper central incisors had not yet been cut. There was no history of paroxysmal hæmoglobinuria; he regarded the latter and Raynaud's disease as different expressions of the same state, and the former, in children, was almost entirely due to congenital syphilis. In all the cases of paroxysmal hæmoglobinuria so far narrated before the Society there was some history of syphilis. A year or two ago, Dr. James Taylor showed a case before the Society in which at the time there was no history of syphilis, but two months afterwards the child developed specific ulceration on the soft palate and uvula. Dr. George Carpenter showed a case in which, though there was no evidence of syphilis at the time, the brother and sister were under his care at hospital with congenital syphilis. He (Dr. Guthrie) referred to two sisters under his care, both of whom had paroxysmal hæmoglobinuria, and with marked stigmata of congenital syphilis.

Dr. Travers Smith (Kilburn) said he had had a case of Raynaud's disease under observation for several years, and had attended the parents, but could not find any evidence or history of syphilis in any member of the family. The father of the child had an ulcer on the outer malleolus, and one on the inner malleolus, but no varicose veins. He had thought the ulcers were due to Raynaud's disease. In Dr. Morison's case there was an ulcer on the ball of the foot. The father of the case he had spoken of had cardiac attacks and great dyspnœa, with almost general anasarca. Rest in bed and treatment by tincture of digitalis resulted in cure. No organic murmur had ever been detected in the case.

Dr. Alexander C. E. Gray (South Kensington) asked whether Dr. Morison had any experience of treatment by the constant current in such cases.

Dr. James Taylor differed from Dr. Guthrie's dictum that cases of Raynaud's disease and paroxysmal hæmoglobinuria were always due to syphilis. He had two cases of Raynaud's disease in the same family now under observation, but there were no

signs of congenital syphilis. The disease had often been seen in adults who had no signs of syphilis.

The Chairman (**Dr. Edmund Cautley**) expressed agreement with Dr. Taylor's remarks. The majority of the cases of the kind had no connection with syphilis, though the latter might lead to local gangrene, such as he had seen in the ears, but the condition cleared up on the administration of anti-syphilitic treatment, and there had been no recurrence.

Dr. Alexander Morison, in reply, said his case had been treated by dieting, warmth, and rest in bed. At first he thought there might be a syphilitic element in the case, and advised mercurial inunction and the administration of small doses of iodide of potassium. It was very difficult to arrive at a positive conclusion as to the invariable origin of such a condition. In the family of the case under discussion, the first child was still-born, the third was prematurely born at the seventh month; and of course the most frequent cause of such happenings was a specific taint. It was difficult to exclude a specific taint. He had personal knowledge of a family for eighteen years, all the children of which, except for some nervousness, were healthy. There had never been any infection of any kind. When the children were almost fully grown the father developed an ulcer on his lower lip, which was cured by iodide of potassium, and he admitted infection twenty-five years previously. The oldest child he had known with paroxysmal hæmoglobinuria was a girl of about 15 years, whose parents he had known a long time, and they appeared to be guiltless of anything specific, but on his previous experience he would not say there was no possibility of it being a remote effect of syphilis. The sclerodermia mentioned by Mr. Pernet was one of the points brought out in the thesis to which he had referred, which was translated by Sir Thomas Barlow. Dr. Theodore Fisher would find a reference in that thesis to such an accident as he described. He could not answer Dr. Travers Smith's question as to the possible cause of the ulcerations in adults to which he had referred.

36. RACHITIS PSEUDO-PARALYTICA HYPOTONIA MUSCULARIS.

By LEONARD G. GUTHRIE, M.D.

The exhibit is a fairly nourished female child, aged 2 years. Its mother says it is as helpless as a new-born

babe. She is the youngest but one of eight, who were born in rapid succession, and who have all suffered from "consumptive bowels." The youngest, aged 6 months, is said to be "going the same way as this." The family live in the country near Maidenhead.

The patient is very undersized, and her weight is 15 pounds. Her intelligence is poor; she cannot speak and looks dull and lethargic.

The head is rachitic in form, with a square and overhanging brow; the anterior fontanelle is open and the size of a shilling. The teeth are normal in number and are sound, with the exception of the upper central incisors which show signs of incipient decay. The thorax is not markedly rickety in appearance, but the costal epiphyses are slightly enlarged, and the costal arch is wide. The abdomen is large and protuberant. The liver and spleen are not enlarged.

The child makes little attempt at voluntary movements, except that of constantly sucking its fingers. It does not try to sit up, but when placed in a sitting position can maintain it, but bow-backed and huddled-up.

When placed on its feet it will not stand, but keeps the thighs flexed on the abdomen, and would collapse if it were not held up. No attempt at walking is made. It does not resist any passive movements and retains any attitude in which it may be placed. It can lie with its head between its fully extended legs, and the lower extremities can be flexed until the feet touch the face. When lying on its back the lower limbs fall outwards in "frog-like" or cadaveric position. The knees can be hyper-extended (genu recurvatum). There is also some genu valgum. There is undue motility of all the joints; the feet and hands can be dorsiflexed until the fingers and toes almost touch the backs of the forearms and fronts of the legs respectively. The epiphyses are not enlarged.

Although voluntary movements are feeble and rare there is no paralyses of any part, but the muscles are small and flabby.

The knee-jerks are normal and the plantar reflexes are flexor in type though sluggish.

The condition is one of muscular "hypotonie" with undue laxity of the articular ligaments, in association with a moderate amount of rickets. It may be regarded as a congenital defect or want of development rather than as an example of ordinary rickets. Perhaps its immaturity may be the result of its parent's prolificacy and poverty.

It was weaned at three months of age, then fed on cow's milk and water, half and half, and subsequently on biscuits, bread and butter, and Benger's food. The mother forgets how much milk it takes "per diem" now, perhaps because the quantity has been small.

Although it lives in the country it has been seldom taken out of doors because the mother has so many other children to look after.

(*Exhibited February the 20th*, 1903.)

Discussion.

Dr. G. A. Sutherland regarded the case as one of rickets pure and simple in which the stress of the disease had fallen especially upon the muscles and ligamentous tissues. The child had had convulsions, and had the appearance of a rickety subject. There was also tetany. Dr. Guthrie had the power of settling the diagnosis, because, if it was rickets, proper feeding would result in a marked increase of power in the muscles and of strength in the ligaments. The condition of hypotonus in infants was more marked in connection with Mongolian children than in any other class that he knew. Still, in rickets there was great flaccidity, and coxa vara was associated with the same condition. He regarded Dr. Guthrie's case as one of pure rickets, rather than one in which there was any particular congenital defect.

The Chairman (**Dr. Edmund Cautley**) said one was apt to attribute to rickets all that a rickety child suffered from. Possibly Dr. Guthrie would say that the reason the cartilages and bones did not show the rickets more markedly was that the child was so ill-developed. On the other hand, it might be suggested that the condition was one of maldevelopment, and that the relaxation of ligaments was due to that maldevelopment rather than to the rickets. Possibly both were due to the same cause. The ligaments were very elongated, and the joints

almost flail-like, and that was likely to be due to maldevelopment, or malnutrition, rather than to the rickets itself.

Dr. Leonard Guthrie, in reply, cordially agreed with Dr. Cautley's remarks; it was rickety, and he showed it as rickets with unusual manifestations. He agreed also with Dr. Sutherland, that under treatment and careful management the child would considerably improve.

37. ON CASES OF UNCOMPLICATED MYOCARDITIS IN CHILDREN.

By GEORGE CARPENTER, M.D.

Myocarditis has been divided into parenchymatous and interstitial, and the lesions encountered are either circumscribed in single or multiple areas or are diffuse in their distribution. Further, the parenchymatous and the interstitial changes in the cardiac walls, although they may occur independently, are not infrequently associated in varying degrees. The degenerative changes in the muscles-fibres, in the shape of albuminous, fatty, and waxy alterations caused by toxin poisoning, are met with in the initial stages of the infectious diseases and occur during convalescence; these also arise from other disorders and are now well known in the post-mortem room, but rarely is it possible to diagnose these changes during life, though they may be suspected. But, parenchymatous myocarditis is not necessarily a fatal complaint, even when the symptoms are extremely well marked, as an illustration of my own, to be related later, will show.

Interstitial myocarditis, which is looked upon as a rare disease, is met with as an acute disorder, or as a chronic complaint of the cardiac wall, which is thought to be rarer still. Of 56 cases of chronic myocarditis collected by Quain, but 3 occurred in children.* The commonly

* Keating's 'Cyclopædia of the Diseases of Children,' vol. ii, part 2, p. 841.

recognised causation of the acute disorder is pyæmia from bone and joint troubles. The common sequence is the formation of an abscess in the heart wall, which either bursts into the pericardium, producing pericarditis, or into the endocardium, and originates an acute cardiac aneurysm. Of 25 cases of abscess of the heart collected by Quain 14 of them occurred under 15 years of age.* On the other hand, pyæmic pericarditis may be associated with, and not be the cause of, interstitial myocarditis.

Acute myocarditis of rheumatic origin must be here mentioned, for although perhaps the changes in the heart muscle are usually more of the nature of parenchymatous degeneration than of interstitial inflammation, nevertheless, these conditions are usually associated. Acute myocarditis has been found in connection with old and with recent valvular disease, and also with pericarditis. It has also been met with in scarlatinal pericarditis.

In regard to chronic interstitial myocarditis, Dr. Theodore Fisher† has called attention to the fact that small fibroid patches are frequently seen scattered through the heart muscle in fatal cases of mitral stenosis. He gives an illustration of fibrous myocarditis in a lad 18 years old, associated with old aortic disease. He also relates a case of an infant 4½ months old who was found post mortem to have fibroid disease of the cardiac muscle and stenosis of the aortic valve. Chronic interstitial myocarditis may also be associated with adherent pericardium, but it is not, however, in every case of hypertrophied heart from adherent pericardium that such changes can be found microscopically. Sclerotic patches in the heart may also take their origin in occlusion of the coronary arteries, syphilitic or otherwise. Syphilitic myocarditis, in the shape of solitary and multiple gummata, has been recorded in infants and children, and Hektoen‡ has published a case of syphilitic interstitial myocarditis in an infant 6 weeks old. Dr. J.

* Ibid., vol. ii, part 2, p. 840.

† 'Bristol Medico-Chirurgical Journal,' March, 1900.

‡ Carpenter, 'Syphilis of Children,' p. 57.

Porter Parkinson * has also shown to the Society a syphilitic nodule in the heart of an infant aged 3 months, which was composed of young inflammatory tissue in various stages of development, the adjacent muscle being infiltrated and destroyed.

Judging from the paucity of the literature on this subject, it is a malady about which little is known, and which therefore requires further attention on the part of those specially studying children's complaints. The following striking examples of so rare an affection as uncomplicated myocarditis appear to me to be of sufficient interest to bring them to the notice of members of this Society, in the hope that by calling attention to myocarditis and to the necessity for combined action our knowledge on this subject may be materially strengthened thereby.

Case 1.—The patient, a girl aged 6 years, was the child of healthy parents, but with a strong family history of phthisis. She had a rash on her buttocks and "snuffles" soon after birth. She had been ailing for a month, her illness commencing with enlarged glands in the neck, and her mother said that she had wasted and suffered from a swollen abdomen and complained of pain at the pit of the stomach. When she was admitted into the Evelina Hospital on September the 3rd her face was dusky and her lips were blue. The cardiac impulse, which was feeble, was felt with difficulty outside the nipple line in the fifth interspace. The dulness commenced at the third left costal cartilage, and extended well to the right of the sternum. A whistling systolic bruit was heard over the cardiac impulse, and a systolic thrill was felt on placing the hand over the cardiac area. There were no adventitious sounds to be heard in the lungs. The abdominal walls were flaccid, and the liver, which was pulsating, reached almost to her umbilicus. The spleen was not enlarged. She passed 18 ounces of urine during the first twenty-four hours; it was of a specific gravity of 1026, and it

* 'Reports of The Society for the Study of Disease in Children,' vol. i, pp. 141, 142.

contained about one third of albumen. The pulse-rate was 132 to the minute, the respiration rate was 56 to the minute, and the temperature was 97·6° F. By the 6th the bruit was not to be heard, and the liver was returning to the costal margin. Her general condition had greatly improved. On the 12th she was still blue round the mouth. The cardiac impulse, which was exaggerated, was felt half an inch outside the nipple line. There was no epigastric impulse, and the area of cardiac dulness was not enlarged to the right. The first sounds at the apex and the left base were reduplicated. The liver was now on a level with the costal margin. The specific gravity of the urine was 1024; 18 ounces were passed, and it contained one eighth albumen; there were no casts. The temperature was still subnormal. The pulse-rate was 100 to the minute, and the respiration-rate was 32 to the minute. On the 15th there was a trace of albumen in the urine, on the 16th one fortieth, on the 17th there was none, and it was subsequently free during her stay in the hospital. On October the 1st she was found to have paralysis of the soft palate and the knee-jerks could not be obtained. By the 22nd food ceased to pass by the nose and the soft palate had in great measure recovered; the knee-jerks were still absent. On November the 8th she got up, and she was discharged on the 11th with a sound heart. Subsequent inquiries directed to her history revealed the fact that three weeks before her admission into the hospital she had "patches" on the throat, for which the medical attendant ordered a paint, but she was not ill enough to be confined to her bed.

Case 2.—The next case is that of a girl aged 9 years, who was admitted into the Evelina Hospital on September the 2nd for a swollen abdomen and shortness of breath. The former trouble was supposed to have started in the preceding January. Her father suffered from heart disease. On admission she was a well-nourished, rosy-cheeked child, and the most striking features of her case were a regurgitant venous pulse in the neck and an

obviously pulsating liver. The pulse-rate was 112 to the minute, the respiration-rate was 46 to the minute, and her temperature was normal. The cardiac impulse was half an inch outside the nipple line; it was not well defined. There was a suspicion of a thrill to be felt there. The area of dulness, which commenced at the left third costal cartilage, extended half an inch to the right of the right sternal margin. There was no bruit. The lungs were normal. The spleen was not enlarged. The free edge of the liver was a hand's breadth below the costal margin in the nipple line. The urine was free from albumen. Whilst under observation she had paroxysmal attacks of facial cyanosis. Sometimes her lips were blue and sometimes the face was flushed. By September the 9th the liver had decreased in size and pulsation was not so marked. Several examinations of the urine had been made prior to this date, but with negative result. On the 11th suddenly the face became very blue, the eyes were fixed and the limbs were rigid, the pulse was imperceptible at the wrist, and she died.

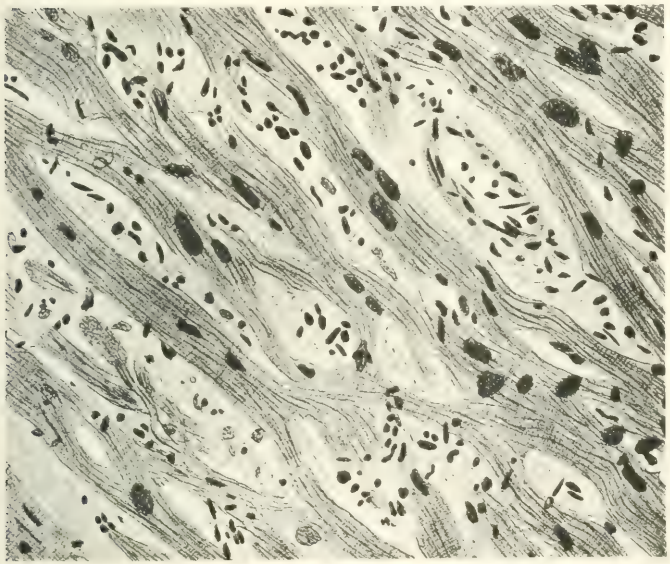
At the post-mortem examination the lungs were found to be very dropsical. The pericardium was healthy. The heart weighed 9 ounces.* The left ventricle was pale, hypertrophied, and showed tabby-cat striation. The left auricle was dilated, thin, and pale. The endocardium was smooth. The mitral valve showed haemorrhages at its root and in the valve itself; it admitted three fingers. The aorta presented patches of atheroma above the left coronary artery. The right ventricle was considerably dilated, pale, and showed tabby-cat striation. The right auricle was also much dilated, and its substance was pale. The liver, which was of the "nutmeg" type, weighed 2 lbs. 14 oz. The kidneys weighed $7\frac{1}{2}$ ounces; they were heavy, hard, and leathery. The glomeruli were injected and the vessels of the medulla stood out prominently. The spleen was not enlarged. The stomach was congested.

Under the microscope the muscular fibres of the heart, for the most part, showed well-marked longitudinal striæ.

* Normal weight about $4\frac{1}{3}$rd ounces.

In places the transverse striæ were well or fairly well marked, but in others they required very careful focussing for their detection. On the other hand, though quite exceptionally, some fibres or portions of a fibre had a ground-glass appearance with obliteration of all striation. Accentuation of longitudinal striation and faintness or absence even of transverse striation were the prevailing characteristics. The muscle corpuscles with but few exceptions stained readily everywhere, and many of them

FIG. 20.



were very large and prominent. Certain microscopical fields showed an abundance of vessels; occasionally a small vein was found between the muscular bundles surrounded by a crowd of white corpuscles. Slight or an easily recognised increase of nucleation of the inter-muscular areolar tissue, with readily detected diapedesis of white corpuscles from the neighbouring capillaries, was noticed throughout, together with proliferated areolar tissue cells; but here, again, areas were found where this

had not occurred, and the nuclei were quite within normal limits. Specimens stained with osmic acid were unaffected. The pericardium and the endocardium were free from abnormality.

In this case the cause of the interstitial myocarditis was not determined; in the next illustration, however, there appears to be no doubt that rheumatism originated the malady.

Case 3.—A girl, aged 8 years, was admitted into the Evelina Hospital on August the 20th. Until two months previously she was perfectly well, but she then developed rheumatism and subsequently suffered from chorea. On admission she had rheumatic synovitis of the left hand and wrist, the left ankle and the back of the corresponding foot, and a slight effusion into the left knee-joint. Her pulse-rate was 120 to the minute, the respiration-rate was 44 to the minute, and the temperature was 100·4° F. She had a loud mitral murmur, which was audible at the angle of the scapula, and a few rhonchi were detected in the lungs. Under salicylate of sodium the temperature had fallen to normal by the 27th. On September the 8th she was quite free from articular pains and fever, but there were great distress of breathing and considerable præcordial pain. Neither the pulse-rate nor the respiration-rate had increased in frequency. The cardiac impulse was much diffused; it was felt in the sixth interspace half an inch outside the nipple line. Systolic and presystolic murmurs were now heard, and a systolic thrill was felt in the fourth left intercostal space. The liver was greatly enlarged, and its lower edge reached as far as the umbilicus. The spleen was not increased in size. The urine was free from albumen. The ankles were œdematous. By the 11th the pulse had assumed a peculiar form; alternate periods of from ten to twenty beats succeeded one another with great regularity. The rate of one set of beats was about 120 to the minute, and the rate of the other set was 60 to the minute. The respiratory movements did not undergo any

corresponding change. She died quite suddenly on the morning of the 12th.

At the necropsy both lungs were extremely œdematous; many old, dry, cheesy deposits were found in the bronchial glands. The pericardium was normal. The heart was much enlarged. The left ventricle and auricle were dilated, and the substance of the former was pale but not friable. The mitral valve, which was free from endocarditis, admitted four fingers. The aortic valves were healthy. Here and there in the substance of the ventricular wall yellowish dots and striæ were seen, and the tips of the papillary muscles, especially those in relation with the mitral valve, were much spotted with yellowish deposits of about the size of a pin's head. An identical condition to the above obtained on the right side of the heart, and here also the endocardium was free from disease. Under the microscope portions of the ventricular walls, taken at random, appeared to be quite healthy. On examining a dotted fragment it was seen that the muscular fibres in certain small areas had entirely disappeared, their place being occupied by countless oil globules of various sizes. Where it existed the change was extreme in degree though markedly circumscribed. The liver was greatly enlarged, it had a thick rounded edge, and was "nutmeg-like" on section. The spleen was normal. The pyramids of the kidneys were intensely congested. The other organs did not present any abnormality.

My next illustration is one of extensive focal myocarditis of a cicatrical nature, and in this case the cause was not quite obvious, though possibly rheumatism played an important part in its genesis. There was no history or evidence of syphilis, but there was a family history of consumption.

Case 4.—A boy aged 12 months was admitted into the Evelina Hospital on February the 12th, for disease of the hip-joint of four months' duration. Four months previously to February the 12th, according to the mother's statement, the back of the left foot, the left hand, and the wrist were

swollen for about three days. During life the heart and lungs were apparently quite healthy. His temperature was above the normal whilst he was under observation, the highest point being 101° F. Forty-eight hours after admission into the hospital he died suddenly.

At the necropsy miliary tubercles were found in the lungs, the liver, and the spleen. The synovial membrane of the hip-joint was thickened, but the bones appeared to be healthy. The pericardium was healthy. The left ventricle of the heart was dilated, and scattered through its wall were a number of white areas, some rounded, others long and narrow, and many of them seemed to pass from the pericardium to the endocardium (Plate I, fig. 1). The muscular substance where not involved looked healthy. There were two or three small rounded spots of infiltration in the substance of the right ventricle. The valves were normal. There was a valvular orifice at the foramen ovale.

On microscopical examination it was seen that areas of new growth invaded and broke up the muscle bundles from the pericardium to the endocardium. They were of varying sizes, and when the microscopic sections were held up to the light it was seen that the outer half of the heart wall had been involved to the greater extent (Plate II). Under the microscope were seen—1. Wavy bundles of fibro-nuclear tissue with elongated wavy nuclei which sometimes invaded the muscle bundles, and separated straggling muscle-fibres from their fellows, and here and there showed dense nuclear aggregation. Some of the muscle-fibres thus isolated showed transverse striation, but in a few this appearance was lost, and they were attenuated and opaque (Plate I, fig. 2). 2. Areas where the connective-tissue fibrillæ were not so marked, though the nuclei were numerous and stained very well indeed. 3. Areas of varying sizes with increase of irregularly shaped nuclei in the intermuscular fibrillæ. The muscle corpuscles in such, in some instances, appeared to be quite large. 4. Areas, though few and far between, of perfectly healthy muscle-fibre and connective tissue. 5. The endocardium much increased in thickness; the peri-

Fig. 1.

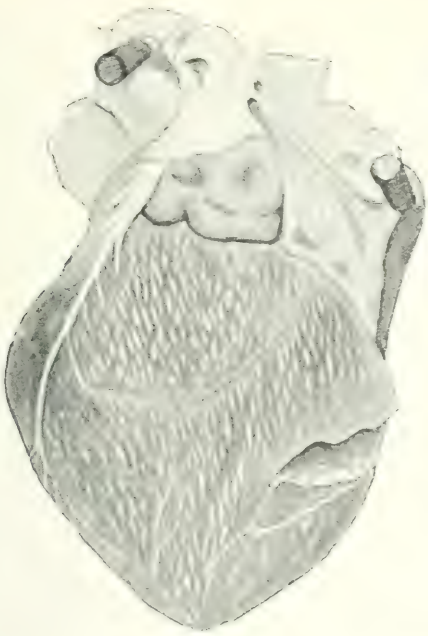
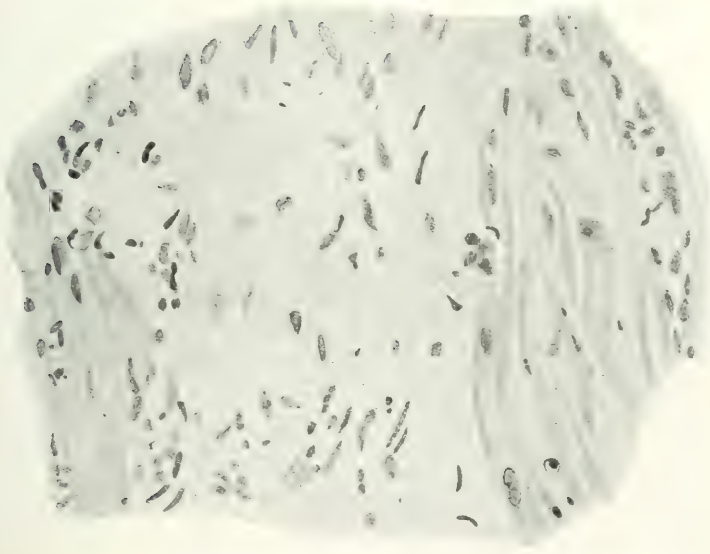


Fig. 2.



Dr. George Carpenter's case of Fibroid Disease of the Heart, in a child 12 months old.

Fig. 1.—The Heart: the Fibroid Areas as they appeared on the surface of the left ventricle and on section of its wall.

Fig. 2.—Microscopic section of a Fibroid Area, showing fibro-nuclear tissue and attenuated muscle-fibres.

cardium, where not joining the sclerosed tissue, unaltered. Although the endocardium was thickened the passage to it of sclerotic offshoots from the neighbouring muscle-fibres was not a feature, but a nuclear increase in these was not uncommon, together with an undue separation of them from one another. The thickening often appeared to be quite independent of, and out of proportion to, any underlying mischief.

In conclusion, I should like to say that I have purposely confined myself to the narration of cases about which there can be no question as to the correctness of the diagnosis, and I have intentionally omitted all reference to clinical cases of myocarditis complicatedwith pericarditis, peri- and endocarditis, adherent pericardium, and cardiac abscesses secondary to pyæmia.

I would also draw attention to the presence of systolic and presystolic murmurs in these free-from-endocarditis cases. It would appear that the mitral murmur merely requires for its production a leaking valve, that no roughness or irregularity of the segments is necessary to that end, and that a mitral murmur is not diagnostic of mitral endocarditis, though it is of mitral insufficiency; that the presystolic murmur and also the diastolic mitral murmur are indicative of a disturbance of the normal relationship that exists between the size of the mitral orifice and the left auricle; that when the latter is distended and the mitral ring is distorted the normal relationship disappears, and leaves in its stead a *comparative* narrrowing of the mitral orifice—in other words, a comparative mitral stenosis for that particular heart; and that murmurs which depend upon these anatomical changes are indistinguishable clinically from the systolic, presystolic and diastolic murmurs of ordinary mitral disease and true mitral stenosis, and that when these murmurs are present they are certainly much more in favour of endocarditis than the reverse by reason of the greater comparative frequency of that condition in heart disease, with the reservation that mitral stenosis in childhood is a rarity. Further, I would

mention that I have heard similar murmurs produced in adherent pericardium associated with a dilated heart free from endocarditis, as was pointed out by Dr. Theodore Fisher,* and in cases of pericarditis and myocarditis in which the endocardium of the valves was healthy.

But, are not the clinical and pathological examples which I have just recorded the key to the explanation of the vanishing mitral murmurs which undoubtedly arise in some cases of acute rheumatism and of chorea, and of the, in consequence, presumed recoveries from endocarditis and from supposed functional cardiac disorders? Do they not suggest that, in some instances at least, the supposed-to-be-present endocarditis did not exist in reality? Do they not invite the conclusion that that which was present was a temporary softening and want of tone of the myocardium, followed by dilatation and subsequent recovery? hence the appearance and disappearance of the mitral murmur. Such a condition of the cardiac muscle would also satisfactorily explain undue rapidity and irregularity of the heart's action in some cases of chorea. It would also explain in these cases of chorea one type of mitral murmur, that which is inconstant in both force and rhythm, and is a not infrequent phenomenon in that disorder, and which entirely disappears by the time the child has recovered or is found to have done so at a later visit. There is one strong point in favour of such a theory in explanation of these conditions, and that is, rheumatic myocarditis undoubtedly exists; we have clinical, supported by pathological, evidence of this. We also know that myocarditis can arise apart from endocarditis.

But, myocarditis is not always attended by a mitral murmur even in an advanced condition of dilatation, nor is it always possible to map out the slightly dilated heart with precision; indeed, the softening might be so slight as to render its physical detection impossible, but, nevertheless, capable of giving rise to definite symptoms. Some such supposition is also reasonable in explanation of the following examples of cardiac disturbance which I have noticed

* 'Bristol Medico-Chirurgical Journal,' 1894; 'Brit. Med. Journ.,' 1894.

in rheumatic fever, and with their recital I will bring this paper to a conclusion.

In one child, aged 9 years, there was extreme weakness of the pulse without obvious lesion of the heart, though the first sound at the apex was almost inaudible. These symptoms lasted for about a fortnight and finally disappeared. In another child, aged 5 years, the pulse was intermittent, and a little later a questionable systolic bruit was detected at the apex, but no further extension occurred, the intermission of the pulse disappeared, and the valvular sound once more became normal.

(*Read February the 20th*, 1903.)

Discussion.

The Chairman (**Dr. Edmund Cautley**) congratulated Dr. Carpenter on the excellent paper he had read and the beauty of the drawings exhibited. The subject was very important, and the condition was frequently overlooked in children.

Dr. Alexander Morison asked if Dr. Carpenter would state what was his experience of the influence of the heart rate upon the condition of the cardiac muscle in children. The only morbid condition he could detect in the drawing submitted was some evidence of defective striation in the muscular fibres. The degree of connective-tissue proliferation was not great. He could not, offhand, diagnose myocarditis from the specimens. Staining with hæmatoxylin gave the connective-tissue colouring in almost any heart. In one of the pictures the longitudinal striation was better seen than the transverse. He would like to hear whether Dr. Carpenter had found the striation indistinct in the majority of the cases he had examined.

Dr. Theodore Fisher (Bristol) urged the importance of microscopically examining such specimens while fresh. In his early days he had a case showing, to the naked eye, most extreme fatty degeneration on the surface of the heart, but after the preparations had been for some time in alcohol very little evidence of the condition could be found. They should be cut at once, or hardened in formalin. Sudan iii is a useful stain. He said that septicæmia was a not infrequent cause of myocarditis. There was some atheroma present in one of Dr. Carpenter's cases. Dr. Mott believed atheroma in children was mostly due to syphilis; but he (Dr. Fisher) thought there was mostly some kind of infection. The matter required further working out. Atheroma was not uncommonly present in cases which had died of diphtheria and typhoid. He (Dr. Fisher)

had been credited with being the first to notice that a presystolic murmur might be associated with cases of heart disease in children in which there was no mitral stenosis. From one or two cases he had seen, he doubted whether the simple explanation given by Dr. Carpenter was sufficient. In one of the cases which he found in Guy's Hospital Reports, in which Dr. Goodhart and other physicians noticed a presystolic murmur, the mitral orifice measured five inches. There could be no doubt that presystolic murmurs occurred far more commonly in children without mitral stenosis than was usually thought.

Dr. George Carpenter, in reply to Dr. Morison, asked that gentleman to re-examine the drawing referred to, for he believed there was a considerable amount of interstitial increase. In the other specimen the sclerosis was so marked that he did not anticipate any difference of opinion.

38. NOTES ON DR. GEORGE CARPENTER'S PAPER CONCERNING CASES OF UNCOMPLICATED MYOCARDITIS IN CHILDREN.

By A. E. SANSOM, M.D.

In an article communicated to this Society ('Reports of The Society for the Study of Diseases in Children,' vol ii, p. 253) I have said that I know very little or even nothing clinically about myocarditis. I do not think that I have to alter this expression of opinion even after the very valuable contribution of Dr. Carpenter, but I do beg to acknowledge with deep thankfulness my indebtedness to Dr. Carpenter for the evidence he has brought forward, and to any observer who brings his mind to bear upon the difficult problems of differentiation which now lie before us.

Now for a critical appreciation of the cases detailed in Dr. Carpenter's communication.

The first case was traceable (happily only traceable) by the clinical history, for there was a recovery, and morbid anatomy did not come in. The physical signs were those of dilated right heart with myocarditis, which probably involved also the musculi of the left ventricle. The true

causes of the myocarditis were in all probability the micro-organisms and toxins of diphtheria.

The second case also showed dilatation of the right heart, but here, alas! morbid anatomy came in to crucially demonstrate the matter. There were signs of septic poisoning of the heart with degeneration of the muscle-fibres. It seems probable that in this case also the myocarditis was brought about by diphtheria.

In the third case no cause seemed to be in evidence other than rheumatism, so far as clinical observations showed.

The autopsy, however, demonstrated many changes of a tuberculous nature, and the evidences of myocarditis might be considered by some observers as more of the nature of tuberculosis than of rheumatism. I cannot doubt that tuberculosis can reinforce rheumatism, and bring about arthritic phenomena which are common to many septic diseases, and it may be a doubtful point whether the signs also observed in the heart in this case, and no doubt of the form of myocarditis, were really the effects of rheumatism or of tuberculosis. Certainly we have no evidence that they could be of the nature of rheumatism uncomplicated and not reinforced by other agencies.

In the fourth case there was abundant evidence of tuberculosis, with changes in the muscle of the heart of the nature of myocarditis. It may be that rheumatism was the prime cause, tuberculosis being the reinforcing agency, or it may be that the changes proceeded alone from the *Bacillus tuberculosis* and its toxins.

So much for the *veræ causæ* of the complex morbid phenomena termed *myocarditis*. I am still of opinion that we have not yet a clear clinical picture.

Then in regard to physical signs, I find myself quite in accord with Dr. Carpenter when he says that a mitral murmur is not diagnostic of mitral endocarditis. It is a common thing nowadays for a consultant to have a case referred to him as one of valvular disease when the question is a doubtful one whether there is any valve alteration at all. If the second sound at the apex preserves

its normal characters it is most probable that, however there may be an inadequacy in the fulfilment of all the details of a normal cardiac cycle, the fault is *not* in a structural change of the valve apparatus.

Even a presystolic murmur, which seems at first investigation to be absolutely indicative of a structural narrowing of the mitral orifice, may be simulated. We have extensive evidence that it can occur in disease of the aortic valves, the mitral being sound. It is true that these cases are rare, and that a well-marked presystolic murmur means *nearly always* mitral stenosis.

It is very probable from recorded evidence that conditions of thickened pericardium with adhesions may produce a presystolic murmur in the absence of the structural lesion of the valve orifice. Dr. Carpenter has adduced further evidence to the effect that in the absence of the structural lesion there may be a diastolic mitral murmur, and from a relative, and not a positive narrowing of the mitral orifice; "in other words, a comparative mitral stenosis in that particular heart." I agree generally with these views, but I acknowledge, as I am sure Dr. Carpenter will do, difficulties. Premising that these cases are rare and very little affect the fundamental doctrine of Gairdner and Fauvel, that a peculiar crescendo murmur occupying the period in the cardiac cycle of the auricular contraction means structural narrowing of the mitral orifice, I will agree that exceptionally they so occur, that they give rise to difficulties, and it is our bounden duty to recognise these difficulties and try to abolish them. In addition to the points urged by Dr. Carpenter I would adduce this difficulty. In cases of mitral stenosis more than in any other cases of valvular lesion I have found a neurosis—the rapid heart, the irregular heart, or the slow heart—come as an epiphenomenon. Now, in some cases it is a question whether the murmur present, which is evidently presystolic, means a permanent structural lesion, or whether it may possibly go away. In the vast majority of cases of course it persists, but I have had instances of its disappearance, and then the question comes,

was it organic at first, or could it be from functional causes? I hold that even a disappearing murmur such as we have noted may be due to structural causes, but that by judicious treatment the passage between auricle and ventricle will become sufficiently large and smooth, so that no murmur nor other evidence may result.

The means whereby this desired result can be attained are pure air, judicious exercises and bathing, and avoidance of rheumatic developments. Medicinally, I think the alkaline bicarbonates, ten grains of each, with aromatic spirit of ammonia twenty minims (and salicylate of sodium at intervals), with tincture of gentian twenty minims, and pure water to an ounce three times a day the best treatment. (*Read February the 20th*, 1903.)

39. A CASE OF CONGENITAL PAROSTEAL SARCOMA ARISING IN CONNECTION WITH THE ACROMION PROCESS OF AN INFANT.

By HENRY J. CURTIS, B.S.

There was no family history of malignant disease or tuberculosis. With regard to the possible influence of traumatism, it may be stated that no instruments were required during delivery, labour being perfectly normal.

At birth, a tumour the size of a small hen's egg was noticed over the left shoulder, the skin covering it being slightly red. The size of the tumour had rapidly increased shortly before the child was admitted into the North-Eastern Hospital for Children, at 5 months old, on December the 31st, 1902, when it was fairly well nourished. Whilst under observation in the hospital for three weeks, the swelling over the left shoulder increased in size by one and a half inches from before backwards, and by one inch in circumference. The temperature had remained practically normal throughout.

The measurements two days before the operation were—Horizontal circumference, 11 inches; from before back, over the top, 9 inches.

The tumour formed for the most part a smooth, rounded swelling, the size and shape of a rather large orange. Somewhat constricted from the main mass at the anterior and inner (median) extremity was a smaller swelling about the size of a walnut, with several smaller rounded or ovoid nodules springing from its outer surface the size of pigeons' eggs. These smaller ovoid nodules felt like very tense cysts, the consistence of the major part of the tumour being firm and elastic. There were three areas, one in front and two on the outer and posterior aspect where definite fluctuation seemed to be present.

The tumour extended from the left clavicle, below and in front, to just above the lobule of the left ear. Anteriorly it extended to the left sterno-mastoid muscle, but was movable over it. Behind and below it descended over the spine of the left scapula to within an inch and a half of the inferior angle. Externally it reached to the outer end of the left clavicle, where it appeared to be attached. It was quite free as regards that bone elsewhere. Actually it proved to be attached to the outer end of the acromion.

The tumour could not be moved over the deep structures apart from the scapula. In the skin over the upper part were several large veins, and there was a small but distinct nævus over the most prominent part of the swelling.

Operation.—On January the 23rd, 1903, under chloroform and then ether, the tumour was readily removed through an incision made from before back over the top of the swelling. The only place where adhesions of any note were met with was at the lower border of the acromion process and the adjacent inferior border of the acromio-clavicular articulation. Three small glands were excised from above the tumour.

Some redundant skin required removal, and a few vessels

of small size were ligatured. The wound was drained at its lower angle, elsewhere being closed.

During the operation though there was comparatively little hæmorrhage there was considerable shock, hypodermic injections of strychnine and ether, and a rectal injection of brandy and saline being found necessary. The rectal injection was repeated after the operation, owing to the very collapsed condition prevailing. On the day after the operation there was free serous discharge through the drainage-tube, which was shortened.

Two days later the drainage-tube was removed, some effused blood-clot being expressed from the wound. A week later all the stitches were removed. The patient left the hospital a week later, on February the 8th, with a perfectly normal temperature. The wound was healed, and all the effused blood had become absorbed; the skin, however, remained slightly discoloured.

On February the 13th, 1903, the child was brought to the hospital in a feeble condition, and a gland an inch and an eighth in length was noticed for the first time in each groin, and another gland about three quarters of an inch long in the right axilla. In the left axilla there was no glandular enlargement evident, and the wound remained soundly healed.

The feeble condition of the child and the enlarged inguinal glands, taken with a rise of temperature reported by the mother to have been persistently above 101° for the previous week, made one suspect the occurrence of general invasion with sarcoma. However, by February the 28th the child's general condition was much improved, and the enlarged inguinal glands had returned to the normal size.

Description of the growth.—The tumour removed was definitely encapsuled, but the capsule was very thin, and almost absent on the deep aspect.

A cut section showed the tumour to be lobulated, of a white, somewhat granular surface, showing small areas of hæmorrhage, especially in places at the periphery. The consistence was distinctly soft and friable, the appearance generally being that of a typical sarcoma.

Microscopically, the growth proved to be a typical, mixed small-spindle and round-celled sarcoma, the areas of small round-celled growth lying between the bundles of well-developed but small spindle-cells, which contained the usual oval-shaped nuclei. There were a considerable number of thin-walled new vessels, and areas of extravasated blood-corpuscles were to be seen of varying size. The small glands removed were remarkably vascular, as seen in stained section, and the loose fatty tissue around was even more congested. There was, however, no evidence of infiltration with growth, and in this opinion I am happy to be confirmed by Mr. A. G. R. Foulerton.

Remarks.—The points of interest in this case are—

1. The question of clinical diagnosis.
2. The remarkable ease with which the tumour shelled out, in spite of its sarcomatous nature, accounted for by—
3. The thin but well-defined capsule, and the ill-defined seat of origin of the swelling, which was, in fact, a *parosteal* growth, as distinguished from a sarcoma originating in the cervical lymphatic glands.
4. Its congenital nature.

1. The diagnosis had firstly to be made between a benign and a malignant tumour. In favour of benign were the congenital origin and the physical signs already mentioned, notably the smaller nodules, feeling like tense cysts, and the fluctuating areas, which strongly suggested the condition to be one of hygroma or lymphangioma, the presence of the distinctly nævoid condition of the skin over the most prominent part further suggesting a mixed angioma and lymphangioma.

The congenital origin, however, in no way contra-indicated malignancy, and in favour of it were the marked fixity of the tumour to the deep structures, so that movement independent of the scapula was impossible; and secondly, the rapid growth since birth, especially during the three or four weeks prior to admission and operation.

2 and 3. The fixity and situation of the growth, which was of very considerable size for a child only five and three-

quarter months old, made one anticipate some difficulty in its removal, which proved, however, to be comparatively easy, no large vessels being involved. The tumour was definitely encapsuled, and was only slightly connected with the spine of the scapula, in all probability arising in the tissues immediately outside the periosteum, thus belonging to the rarer variety of peripheral (as distinguished from endosteal) sarcomata, and usually designated as *parosteal*, which, as Clutton * remarks, only secondarily invade bone.

Erichsen refers to what is apparently an exactly similar case in his own practice, occurring in an adult, and from the account, which may be conveniently quoted in detail, it would appear to have been parosteal at its commencement, only secondarily invading the spine of the scapula. "A tumour as large as a full-sized turnip was removed from the shoulder of a middle-aged man, and was found to be slightly connected with the spine of the scapula. It presented all the characters of a spindle-celled sarcoma, consisting almost entirely of densely packed, fusiform cells, with oval or oat-shaped nuclei. A small mass reappeared before the wound had completely healed. It recurred a second time, and a portion of the spine of the scapula, removed with the tumour, showed that the growth had sprung from the cancellous tissue." †

With regard to the actual situation of the growth, Bland-Sutton, in his work on 'Tumours,' speaking generally (not especially referring to children), says that the scapula is sometimes attacked by sarcoma, usually springing from the body of the bone, the coracoid process exceptionally being the seat of origin. He speaks of sarcoma of the clavicle as being excessively rare. This is of some interest in the present case, as the slight connection of the tumour with the acromion was close to the outer end of that process, and therefore immediately adjacent to the lower margin of the acromio-clavicular articulation.

Its parosteal origin is largely confirmed by the microsco-

* Treves's 'System of Surgery,' vol. i, p. 914.

† 'Erichsen's Surgery,' vol. i, p. 1039.

pical appearance, spindle-celled sarcomata arising primarily especially in the periosteum and tissues in its neighbourhood, in fasciæ, and in the secreting glands—kidney, ovary, testis, parotid, etc.

As a few small glands were also removed from above the growth, it may be as well to state that primary sarcoma of the cervical lymphatic glands, as Pearce Gould says,* is not a common disease, is generally met with in adults at or past middle life, and consists microscopically of small round-cells embedded in a very fine wide-meshed stroma.

4. The congenital origin of this sarcomatous tumour in such a situation as the shoulder, as distinguished from the internal organs, is of some interest. According to Ashby and Wright,† the connective-tissue group of tumours is that almost exclusively met with in children. Sarcomata appearing after birth are "rare in children, are most often seen in connection with the periosteum, and often follow injuries." The same authors also record a case of rapidly growing sarcoma as a sequel of acute periostitis.

With regard to congenital sarcoma of the viscera, the kidney is perhaps the organ most frequently involved, and F. T. Paul is of opinion that renal sarcomata are probably invariably of congenital origin.‡ The present writer recently had to perform hysterectomy in a child of five weeks for sarcoma apparently arising from the posterior lip of the cervix uteri, fungating into, and infiltrating the wall of the vagina.§

With regard to congenital sarcoma, apart from internal organs, the present writer has seen one other case, operated on by Mr. Russell Howard in connection with the periosteum of the tibia in a child five weeks old. The growth formed an ill-defined and somewhat diffluent swel-

* 'International Text-book of Surgery,' vol. ii.

† 'Diseases of Children,' p. 764. (See also a paper by Dr. Hawthorne and the present writer, 'Transactions of The Society for the Study of Disease in Children,' vol. ii.)

‡ Ibid.

§ This case is also quoted by Lewers in his work on 'Cancer of the Uterus.'

ling just above and behind the base of the internal malleolus of the right tibia. It was about the size and shape of a chestnut, and its consistence was such as to suggest an hygroma. The microscopic appearance was reported to be that of a round-celled sarcoma with fibrous stroma.

According to Mr. Stansfield Collier, however, tumours with such an appearance microscopically, when occurring in young children sometimes show an extremely low degree of malignancy. They recur very slowly, and grow very slowly, lasting for years. One such case, still alive, he has had under observation for at least four and a half years, and he prefers to look upon this class of growths as fibro-cellular, rather than as being sarcomata in the usual sense of the word. This type should therefore be remembered in attempting to make a prognosis.

(*Exhibited March the 20th*, 1903.)

Postscript (September, 1903).—The child was seen recently and remains in good health.

40. A CASE WITH CONGENITAL DIMPLES, COMPRESSION MARKS IN THE SKULL AND LIMBS, AND CLUBBED FEET.

By H. STANSFIELD COLLIER.

On behalf of Mr. Stansfield Collier, Mr. H. J. Curtis showed an 8 months' old boy, born prematurely at 8 months, the membranes having also ruptured a fortnight previously, so that labour was unusually dry.

There was extreme dimpling of the skin over the olecranon processes and both sides of the knees. Both upper and lower limbs, particularly the legs and thighs, showed very marked lateral compression, also seen in the skull, the forehead being in addition flattened from before back, and the tip of the nose depressed, as is so frequently observed after the cure of a severe form of harelip.

A vertical groove existed over the middle of the body of the right inferior maxilla, as also at the postero-internal aspect of the right leg in its lower third. This groove in the leg, taken with a slight one surrounding the remarkably prominent and pendulous buttocks, it was suggested might be accounted for as the result of the funis constricting the buttocks and then passing down along the back of the leg.

Both feet were clubbed, the right in a position of talipes equino-varus, so fitting accurately, as so frequently happens, into the sole of the left which was in a condition of calcaneo-valgus. A slight degree of skin dimpling is not uncommon. It is probably the result of amniotic adhesions occurring when there is deficient liquor amnii.

In the present instance, the extreme degree and universality of the compression effects would imply very considerable deficiency in the quantity of amniotic fluid, long anterior to the rupture of the membranes a fortnight prior to the premature delivery at eight months.

One further point of interest in the case was the evidently stunted condition of the limb bones, especially of the arms and forearms, and of the legs to a less extent, when contrasted with the body length, the finger-tips, for instance, barely reaching to the iliac crests.

(*Exhibited March the 20th*, 1903.)

41. A CASE OF INFANTILISM IN A BOY AGED TEN AND A HALF YEARS.

By G. A. SUTHERLAND, M.D.

The boy was 10½ years old, weighed 28 lbs., and was 38⅝ths inches high. The mother said that at the age of 3 years he weighed 9 lbs. He seemed well formed, and no marked physical defects were obvious. The hands were somewhat blue and the finger-tips a little clubbed, suggesting that some congenital defect of the heart might be present,

though he could not detect anything abnormal. He thought the infantile appearance of the face was well preserved. The mother described his actions as being very like those of a monkey, and stated that when she was six months pregnant she had a fright through a monkey, and soon after the child was born some friends remarked that it looked rather like a monkey! When the child learned to walk it was said to have commenced by leaping, while his method of taking food was very much like that of a monkey. The boy was mentally backward. He had not yet learned his alphabet, and he was four years old before he could talk at all. His voice was husky. His habits were mischievous and destructive. When he was sent to school the teacher reported that he was spiteful. His memory was only fair. The mother believed that he had not grown at all during the last three years. He was not aware that the child had yet been treated, but he intended to put him upon thyroid gland. The boy did not appear to have been specially delicate in the early years of his life.

Exhibited March the 20th, 1903.

Discussion.

Dr. A. E. Sansom said he was glad to hear Dr. Sutherland say he did not think congenital heart disease was excluded because there were no marked signs of that present. The history showed very marked backwardness, and mentally the child seemed very *minus* indeed. What would be the nature of any possible cardiac anomaly? There was no justification in thinking of anything but an open foramen ovale, but that might not show itself by any physical sign. He wished to thank Dr. Sutherland for bringing forward the case, and for what he said about the condition. There might be considerable arrest of development in the heart, and yet there need not necessarily be any physical signs to betray it. The backwardness and the cyanosis might be sufficient to justify an opinion that there might be a defect in the heart.

Dr. George E. Shuttleworth (Richmond) said he was glad to have met the boy again. He discovered him a few weeks ago among some children who were brought for admission to a special school in the Willesden district. The mother stated she had had many offers for the custody of the child, among others

from Barnum and Bailey. He thought that the boy had sufficient mental faculty to start training upon, and that many of his mischievous tricks would disappear when he was taught to use his hands for useful purposes. There were no marked characteristics of cretinism about the case. He certainly thought thyroid administration was applicable to the case, and he hoped to see how the patient progressed under Dr. Sutherland's treatment.

Dr. G. A. Sutherland, in reply, said the doctor who attended the confinement remarked that the child was a midget, but it was not weighed. The mother was not a small woman, but she was said to have had a previous child which weighed 2 lbs. at birth and lived for five months, though it was a seven-months child.

42. HYPERTROPHY OF THE LEFT SIDE OF THE BODY IN AN INFANT.

By ROBERT HUTCHISON, M.D.

The case exhibited was a male baby, 1 month old, suffering from hypertrophy of the left side of the body. The child had been brought to hospital for enlargement and protrusion of the tongue. He was the first child, born at full time, and the labour was natural. There had been no miscarriages. The tongue protruded from the mouth, but the left side was obviously larger than the right, and firm and fleshy, suggesting hypertrophy. Measurements showed that the left side of the chest had a circumference $\frac{3}{4}$ths of an inch greater than the right, and the left side of the head 1 inch greater than the right. The left forearm at its thickest part was $\frac{1}{2}$ an inch larger than the right, and the left leg exceeded the girth of the right by a similar degree. The left leg and arm were also longer than the corresponding right limb by fully $\frac{1}{2}$ an inch. The face presented no visible difference in the two sides. There was no other abnormality.

The case was evidently one of hemi-hypertrophy of the left side, and not hemiatrophy of the right, because of the condition of the tongue.

(*Exhibited March the 20th*, 1903.)

Discussion.

The Chairman (**Dr. Henry Ashby**) (Manchester) said the point which occurred to him during the description was as to how one could distinguish between hemi-hypertrophy of the right side and hemiatrophy of the left. He had been carefully watching two cases recently, one a girl with a history of torticollis and sterno-mastoid tumour. She was now seven or eight years old. The torticollis had been relieved by operation, yet the right side of the face was distinctly smaller than the left. It was an interesting question why sterno-mastoid laceration at birth should give rise to restricted growth on that side as time went on. The other case was one of facial hemiatrophy, or, as Gowers called it, pantrophy, in which the left sides of the jaw, soft palate, and tongue were smaller than the right. There were sclerodermic patches on the right side and on the back. It would be interesting to find some reason for that difference in rate of growth of two sides.

Mr. H. J. Curtis suggested that the plan adopted by Mr. Barker, of taking out a wedge-shaped mass from the tongue and bringing the parts together, should be employed. It was better than using the cautery, perfect union being obtained.

Dr. Robert Hutchison, in reply, said that it was clear that it was hypertrophy of one side and not atrophy of the other, because the tongue was a guide; that was distinctly hypertrophied on the left side. The tongue had not yet interfered with sucking or with respiration, and he thought of leaving it alone until the child was older. If it should need operation, the form mentioned by Mr. Curtis seemed to commend itself, on the assumption that it was purely a hemi-hypertrophy; but if it were a lymphangioma that would be contra-indicated.

43. A CASE OF CHRONIC MANIA (?) IN A CHILD.

By ROBERT HUTCHISON, M.D.

The patient, a girl 5 years of age, exhibited restlessness, incoherence, and excitability. The child's mother was sure that up till three years of age the girl had been perfectly natural, and had walked and talked early. The present symptoms had come on two years before, about the time when the second child of the family was born. The

patient then became passionate, noisy, and excitable, symptoms which had continued, but with diminished intensity, until the present. There was no family history of insanity, and the mother's pregnancy and labour when the patient was born had been normal. The patient presented no sign of any organic disease, and the head exhibited no abnormality. She was very excitable, constantly moving about, laying hold of things in the room. She chattered incessantly, and exhibited a great tendency to repeat questions put to her. She could spell, but not read, and she liked and understood pictures. She was also very sensitive to music—abnormally so the mother thought, and music always excited her greatly. Her memory was good, and she was clean in her habits and slept well. She had never been to school.

On the strength of the history he was inclined to regard the case as one of chronic mania, *i. e.* insanity, rather than as one of imbecility. (*Exhibited March the 20th*, 1903.)

Discussion.

Dr. George E. Shuttleworth (Richmond) said he did not understand why a child who, during the first few years of life, appeared quite normal should suddenly become abnormal, especially at the time the mother was laid aside, unless there were some definite reason. He suggested that the child might have had a fall, setting up some meningitis, without the mother knowing it. The case seemed on the border-line between imbecility and insanity, but seemed rather to belong to the latter category.

Dr. G. A. Sutherland said he saw, a year ago, a child of about seven years of age, the mother of whom gave a similar history to that related by Dr. Hutchison; it was quite well up to the third year, had walked and talked, but that it afterwards became quite imbecile. The simplest course was to doubt the mother's story, because during the first few years of life mothers were apt to exaggerate the mental powers of their offspring, and not until the children were older were mothers convinced of their backwardness. The mother of Dr. Hutchison's case said the child was always very excitable, and he thought possibly the child was not sound during the first few years of life, but that the mother had not noticed the fact.

Dr. Robert Hutchison, in reply, said he highly valued Dr. Shuttleworth's opinion of the excitable child. He admitted that

one must not be too much guided by the mother's story. The tendency was to class such cases as imbecile, but it must be conceded that insanity did occur in children; and a child which was excitable all its life was likely to lose its mental balance, and the advent of another child, and consequent jealousy, might be the determining fact.

44. ACUTE AMAUROSIS FOLLOWING INFANTILE CONVULSIONS.

By HENRY ASHBY, M.D.,
AND
SYDNEY STEPHENSON, C.M.

One of the present writers (1) has recorded a number of cases of amaurosis in infants, in which the blindness was due to attacks of posterior basal meningitis ending in recovery.

We propose to bring before the Society on this occasion another group of cases of amaurosis occurring in early life, which we believe are entirely distinct from the post-meningitis group, and which owe their origin to severe convulsions.

It is not uncommon in the out-patient room to get a history from a mother that her infant or child was left blind for some weeks or longer after having had a severe series of convulsions. We may get such a history when investigating cases of "infantile hemiplegia." The story runs that the child was convulsed for some hours, and when he became conscious again he was blind, and that his arm and leg were paralysed. The sight completely returned, but not the power in the limbs. In other cases the paralysis had been but slight, and the paresis disappeared before the amaurosis.

No doubt much caution must be exercised in drawing conclusions from the history given by friends as to the condition of the infant's sight. Such unskilful observers as the parents of out-patients usually are will be likely to confuse "unconsciousness" or "mind-blindness" with loss

of sight. Moreover, the blindness spoken of might be due to posterior basal meningitis or to a temporary papillitis. But, after putting on one side all sources of error and excluding doubtful cases, there remains a group in which a more or less temporary blindness is "post-eclampsic,"—that is, follows as the result of a series of convulsions with coma, which remind us of the *status epilepticus* of adults. The amaurosis may be accompanied by aphasia and hemiplegia of a temporary or more lasting type.

It may be objected that convulsions do not constitute a "self-standing" disease, but are only accidental complications of many pathological states, and this is no doubt true; but our point is, that whatever their origin, the convulsions themselves are the actual cause of the amaurosis to which we refer. The explosions or nerve-storms may involve the visual cortical centres as well as the Rolandic motor centres, or the speech centre, and the period of discharge be followed by a period of exhaustion. The following cases will illustrate our meaning.

Case 1.—S. R—, aged 3 years, was admitted into the Manchester Children's Hospital on December the 19th, 1902. The mother's history was kindly supplemented by Dr. J. M. Cruickshank, who attended the child during the acute attack. A paternal uncle suffers from epilepsy, and an infant brother died in convulsions. The patient suffered from convulsions a year ago; he has also suffered from eczema. Two weeks before admission, when in apparent health and without any assigned cause, the child was seized with severe general convulsions, which lasted four or five hours. There was drowsiness for several days after the convulsive stage was over. The temperature during the "status eclampticus" rose to 103·5° F.; the pupils were at first pin-head size, but later were dilated and sluggish. Examination of the eyes by Dr. Cruickshank showed no optic neuritis or any coarse changes; there was no albumen in the urine. One of the first things the child said when coming round was, "Mummy, draw the blind up," and his

repeating this request several times called the attention of the friends to the fact that he could not see.* There was some paresis of the left arm.

On admission it was noted there was no paresis; the knee-jerks and plantar reflexes were normal. The boy seemed completely blind, but the pupils were of the normal size and acted to light. The pupils contracted well when light was thrown on them with an ophthalmoscopic mirror, but the child did not seem to notice the light. They dilated well with atropine. The fundi were normal. His hearing was abnormally acute; he would sit up in bed playing with toy animals, which he recognised by touch, and chatting to the other children when he heard their voices. He would sing nursery rhymes and accompany on a toy piano, but often missed the keys, as he could not see them. About ten days after admission, when the ward was lighted up at night, he repeated, for the first time, "Light! light!" and about the same time he was conscious of the light of a taper when brought near his face, and when asked to blow it out he tried to do this, but usually in the wrong direction. He could recognise a watch, if placed in his hands, by putting it to his ear, but he could not see it. By January the 16th it was evident his sight was returning; he could recognise objects, such as books or a watch, a couple of yards off. After this there was rapid improvement, and by January the 29th he could recognise toys on the other side of the ward and pick up beads and pins. He was clumsy for a while on his legs, and he ran against things and knocked his head against chairs for some time after it was certain that he could see. Recovery appeared to take place in both eyes slowly and equally, and we could obtain no evidence of hemianopia or that central sight returned before peripheral, but the fact that he knocked his head against the ward furniture when running about was suggestive of his peripheral vision being less acute than the central.

* It is interesting to note that the child had a clearly pictured memory-image of the window with the blind up, and also of the state of darkness when the blind was down.

Case 2.—George T—, aged 7 months, was first seen on December the 1st, 1897, at the North-Eastern Hospital for Children, London. He had been wasting for a fortnight, with occasional vomiting and "constipated bowels." He had been brought up on the breast, and when examined had four teeth. *January the 26th*, 1898.—Since the last visit he has had "congestion of the lungs," followed by fits lasting for two weeks. The baby was found to be blind after the fits. There is now weakness of the right arm and leg. No optic neuritis or coarse changes in the fundus can be detected. The pupils are equal and active. There was no family history of fits, or lunacy, or of epilepsy. *February the 23rd.*—The child still appears to be blind. *March the 9th.*—Some vomiting still occurs from time to time. The eyes do not follow a light. The pupils are active. There is no rigidity of the right arm and leg, both of which he is able to move slightly. *May the 11th.*—The mother is certain that the child is blind. There is slight rigidity of the right arm and weakness of the right leg. The knee-jerk on the right side is somewhat *plus*. The optic discs were thought to be slightly white, but the ophthalmoscopic examination was difficult, incomplete, and probably fallacious. It appears that the baby had several fits a week or so since, and the last fit was followed by a period of apparent unconsciousness. *June the 15th.*—The child now moves all his limbs freely, and no rigidity can be made out. The knee-jerks are exaggerated. The eyes follow the movements of the ophthalmoscopic mirror. *September the 7th.*—About a month ago the mother noticed that the child looked at things, and she is of opinion that he can now see with his right but not with his left eye. *February the 25th*, 1903.—The child is now nearly six years of age. The mother states that the sight slowly improved until it became quite good. The right arm and leg also recovered. He was unable to walk until six months since, but now gets about nicely by himself. Sight is good; the fundi are normal. The knee-jerks are normal. Intelligence is good. The limbs are of equal girth and warmth.

Case 3.—Alfred P—, aged 18 months, was seized with convulsions about two months before he came under care, and they were followed by a period of unconsciousness, which lasted four days. There was intercurrent vomiting. Upon recovery he was found to be blind, and his right arm and leg "hung as if paralysed." The sight is stated slowly to have improved, so that when I saw the case I could not satisfy myself there was any departure from normal in that respect. The pupils were equal and active; the fundi showed no morbid changes. The right arm and leg, however, were distinctly weaker and colder (?) than those of the opposite side.

Case 4.—Annie S—, aged 14 months, had suffered from a cerebral attack some four weeks before she fell under notice. This was marked by convulsions, vomiting, semi-consciousness, "screaming fits," and incontinence of urine and fæces. The symptoms persisted for four or five days, and then gradually passed away, when it was found that the child was perfectly blind. The right leg was also weak. When the baby was seen she did not follow the flash of the ophthalmoscopic mirror at all readily, and the vision of the right was thought worse than that of the left eye. Her sight, by the way, was stated to be improving. The pupils and fundi (the latter examined under chloroform) were normal. The case then passed from under observation.

Case 5.—Roland H. T—, aged 5 years, was admitted into the Manchester Children's Hospital on October the 2nd, 1902, with infantile hemiplegia. His mother states that when eighteen months old he had a severe attack of whooping-cough, followed by convulsions. He was convulsed for four hours; this was followed by stupor for some days. It was then noted that his right arm and leg were paralysed, and also the right side of his face. He was blind, and could not speak. His sight gradually returned in the course of a week or two, but he did not speak for a month. He has a well-marked spastic hemiplegia and slight facial paralysis. His intelligence is fairly good.

Remarks.—Nettleship (2) has reported two cases similar to the above, and Gay (3) reports four more. Both authors relate other cases of amaurosis, which are, however, clearly not on all-fours with ours.

If we analyse these 11 cases, *i. e.*, which include our own and those of the above-named authors, we find, as far as age is concerned, 2 were between six weeks and two months, 2 between seven months and eight months; 4 from thirteen months to eighteen months, and 3 from two and a half months to three years. In 2 cases the convulsions occurred during whooping-cough, and one followed "congestion of the lungs;" in 8 cases no cause could be assigned. In one case the convulsions were only slight; in the rest the attacks were evidently severe, the "status eclampticus" lasting for some hours, and unconsciousness or stupor for some days. In one case, aged two months, the convulsions and stupor lasted for two weeks, the amaurosis which followed was still present when seen at seventeen months of age, and there was apparently some optic atrophy, as the "discs were of a light tan shade."

In 4 of the cases there was no accompanying paralysis. In 7 (or 63·6 per cent.) there was more or less paralysis; in one of these the paresis of the left arm disappeared in a week or two. In 4 there was a well-marked right hemiplegia which lasted for some months, and in some of the cases the paralysis seemed likely to be permanent. In one case there was a left hemiplegia, and in another weakness of the legs. The fundi were normal in all but two; in the latter one disc was whiter than normal, and in the other the discs were of a light tan shade. In all except one there was recovery of sight; in the exceptional case blindness seemed likely to be permanent. For the most part the pupils were observed to be normal and active, in others sluggish. In one case there was temporary aphasia. In only one have we a note of the urine, *viz.*, Case 1; in this case there was no albuminuria.

What was the nature and cause of the convulsive attacks? The history in nearly all of the cases records that

the fits were frequent, and accompanied by coma or stupor which lasted some hours or days. Indeed this condition of coma was so serious in some instances that death seemed imminent. In two cases whooping-cough appears to have been the exciting cause; in another "congestion of the lungs;" while in other cases no cause could be assigned. It is possible the toxin of pneumonia or influenza may have been the immediate cause in some of the cases, or, less likely, the convulsions were uræmic. This condition of "status eclampticus" which occurs in infants and young children is in some ways remarkable, and no doubt does duty on occasion for "brain fever" or "meningitis" from which recovery takes place. The post-mortem findings in such cases, according to our experience, are disappointing, nothing but a wet brain with venous congestion being found, at least to the naked eye. Probably this condition is much oftener due to reflex irritation than to central lesions. In those cases associated with a hemiplegia which becomes permanent, there can be little doubt there is a grave organic lesion, such as a thrombosis or hæmorrhage involving part of the Rolandic area or its "hinterland," but whether the lesion is the cause or consequence of the convulsions in any given case must at present be left an open question. The stage of coma, which is so often prolonged for some time after the convulsions have ceased, may be the result of exhaustion or of toxin poisoning, and when consciousness returns the visual centres are left in an anæsthetic state, or the motor speech centre or other motor centres may be left in a temporarily exhausted condition.

There is some evidence to show that the visual cortical centres are involved in the epileptic attacks of adults. It is well known that visual auræ are not uncommon. Gowers (4) records that "loss of sight preceded loss of consciousness in 26 cases out of 1000 cases collected by him. In one case the loss of sight in minor attacks lasted for an hour, and was followed by an hour's sleep. Gowers concludes that "the phenomena of these auræ suggest

that there may be both a discharge and an inhibition of the same centre. For instance, a patient always first saw a flash of light, then sight was lost, and then consciousness."

Auditory warnings as well as visual appear to occur in the epileptic attacks of adults, although perhaps not as frequently. We are not aware of any loss of hearing following the fits of infants or children.

In the "night terrors" as well as in the rarer "day terrors" of children (conditions which are allied to epilepsy) the frights are occasioned by the involutary recall of visual memory-images; the child sees pursuing wild beasts, rats, or other horrors, but does not appear to hear any sounds.

Transient hemianopia, or, at any rate, partial blindness, is apt to occur in association with migraine in neurotic subjects. A neurotic girl under our observation, who often has severe headaches, frequently complains in these attacks that she cannot see anything situated in the right half of the field of vision. In looking at the word "Monday," for instance, she sees "Mon" but not "day."

Dr. W. Harris (5) has recorded some cases of hemianopia following unilateral convulsions in adults with general paralysis, and also following attacks of migraine. He says that "in many cases an epileptic discharge may originate in or near the half vision centre on one side, in some cases proceeding no further beyond producing temporary hemianopia, and in others producing a typical epileptic fit; and again in others giving rise to unilateral convulsions without loss of consciousness."

Dr. F. E. Batten tells us he has also seen a temporary hemianopia follow an epileptic or convulsive attack in a child.

A transient aphasia may also occur in association with a temporary amaurosis following convulsions, as in Case 5. In another case coming under our notice, an aphasia which lasted two months followed a convulsive attack, but was not associated with any blindness. A boy of two and a half years old suffering from whooping-cough was seized with convulsions

which lasted four hours, and when consciousness returned it was found he could not speak. When seen shortly after he appeared quite well, and understood what was said to him, but refused to say a word. He pushed his plate at dinner-time when he wanted more. Two months later he began to say a few words, and then quickly became as big a chatter-box as ever.

We have already noted the occurrence of a temporary paresis of hemiplegic distribution which is found not infrequently after convulsions, recovery taking place in the course of a few days or weeks. This also follows epileptic attacks, as is well known. In infantile convulsions from reflex excitation the spasms are often one-sided, or at least more marked on one side than the other, and the side which has suffered most is apt to be weak for a while.

In conclusion we would venture to suggest—

1. That there is a form of amaurosis which occurs in infants or young children which is post-eclampsic, due to anæsthesia of the visual centres.

2. That the convulsions, which may be due to various causes, are apt to be severe and accompanied by coma.

3. That the amaurosis may be associated with aphasia, and paresis of hemiplegic distribution. The hemiplegia may be permanent.

4. That the amaurosis is for the most part transient. It is possible that in some instances there is hemianopia.

References.

1. "Fleeting Amaurosis in Infants," by Sydney Stephenson, C.M., 'Reports of The Society for the Study of Disease in Children,' 1902.

2. "Recovery from Amaurosis in Young Infants," by E. Nettleship, F.R.C.S., 'Transactions of the Ophthalmological Society,' vol. xiv, 1884.

3. "Acute Cerebral Amaurosis of Infancy," by W. Gay, M.D., 'The Royal London Ophthalmic Reports,' vol. xiii, 1893.

4. 'Epilepsy and other Convulsive Diseases,' by W. R. Gowers, M.D., F.R.S.

5. "Hemianopia, with Special Reference to its Transient Varieties," by W. Harris, M.D., 'Brain,' vol. xx, 1897.

(*Read March the 20th*, 1903.

Discussion.

Dr. A. E. Sansom thought that the part of the paper which was not the least interesting was that which appealed to the imagination, and the element of imagination was largely necessary in the matter of children's diseases. Amaurosis had been defined as a disease in which the patient did not see anything, nor the doctor either. The cause of the amaurosis in such cases as those narrated was not known; it was probably either a poison of the nervous mechanisms involved, or a vaso-motor ischæmia. It might be a local poison, which might have as its expression a neuritis or neurosis. It was either an abnormal condition of the nerve mechanism, or an abnormal condition of the vessels which nourished that mechanism. If it was an ischæmia involving the vaso-motor mechanism, it must involve it more on one side than the other, and if so it threw a flood of light on the very interesting question of the cause of increased blood-pressure. Contractions of arterioles might be regional, and not universal. The contraction was a very difficult question to face. It had been said that alcohol relaxed the arterioles, but it was not so. Alcohol produced tonic contraction of them for a longer period than perhaps anything. Chloroform and all the anæsthetics would contract for a limited time, but dilatation rapidly followed. The fact remained that the contractions might be local and not general, and they might be a reinforcing cause of high arterial tension. There might be ischæmia, which might be in a limited area of the brain or heart or various organs of the body. It was a great encouragement to have cases so carefully detailed as in the paper, and in the case of children there was less likely to be complicating conditions than in adults.

Dr. Robert Hutchison said that a very similar condition of amaurosis could be seen in other cerebral conditions. He had seen it after superficial meningitis, the blindness being transitory, and unaccompanied by fundal changes.

Dr. George Carpenter said Dr. Henry Ashby and Mr. Sydney Stephenson in their paper placed on record what they considered to be a variety of amaurosis in early life distinct from that due to post-basal meningitis. In some cases hemiplegia followed convulsions, together with blindness, the sight returning, but not the paresis in the limbs. In other cases paralysis was slight, and the paresis disappeared before the amaurosis. The condition they described reminded them of the status epilepticus

of adults, a state of exhaustion of cortical centres; and their point was that whatever their origin the convulsions were the actual cause of the amaurosis to which they referred. Quite apart from posterior basal meningitis, he had seen temporary amaurosis in sequence to a series of infantile convulsions in a child eighteen months old who had been subject to eclampsia, and he had also seen it in other conditions associated with convulsions, which he should have ascribed to meningitis rather than to a condition of status epilepticus. The theory which they put forward was certainly a plausible one, but it was a theory, and lacked *post-mortem* confirmation. He confessed that certain of their cases impressed him as being not altogether free from a strong suspicion of meningitis, and in one or two instances the possibility of an intra-cranial hæmorrhage could not be altogether excluded. That a child recovers sight is no proof that serious organic mischief was not present, and to which the amaurosis was due; indeed, sight may be recovered and lost, and recovered and lost again, and yet extensive disease of the brain be present the whole time. The persistence of hemiplegia was, he thought, a strong point against a functional cortical disorder. Neither was the absence of optic neuritis, nor a state of activity of the pupils, a guarantee that there was no meningitis present. Perhaps a case which was under his observation for several years would best illustrate the difficulties and the danger of drawing conclusions as to the absence of organic mischief. It was not quite on a par with the cases narrated by them by reason of the absence of initial convulsions; but it was of interest from the point of view that had the patient not been kept under observation for a very long period it might have been thought to be of a functional character, and it might have been recorded as a case of recovery from functional amaurosis. The patient, a girl, was first seen in December, 1886, with paralysis of the right arm, and possibly of the face, of a week's duration, which had come on quite insidiously. Next she dragged her leg, and in a fortnight from the commencement of the paralysis she had hemiplegia, with knee and ankle-clonus of that side. There were no constitutional symptoms, the temperature was not raised, and the fundi oculorum were quite natural. In March she developed hemichorea, and lost her sight. There were no fundal changes, and she was thought to be suffering from functional amblyopia by the ophthalmic surgeon who saw her at the time. In view of this diagnosis she was looked upon with suspicion, and it was thought to be a case of hysteria. About this time her arm became very painful, and the twitchings in the limb were so severe that it had to be strapped to her side. In June the *left* arm was occasionally rigid, and it was sometimes "jerked about;" the patellar reflexes were exaggerated, and clonuses were obtained to a less extent in the left leg. The

left angle of the mouth was drawn to this side more than to the right. By September she had better control of her legs, and there was but slight if any rigidity. The movements of the left arm on attempting any voluntary exercise were increased in violence, and the inco-ordination and rigidity were more marked. The right arm was about the same. In December, 1887, the sight of the right eye was much impaired, and as it improved that of the left failed. From May to December, 1888, her sight was considered to be normal; but in this latter month the vision of both eyes began to fail again, and she was unable to read or to sew. In February, 1889, the vision of each eye was $\frac{6}{60}$ths, central vision was defective, and the field was limited. The left optic disc was certainly normal in appearance, but the right optic disc presented rather small and somewhat tortuous arteries, and it appeared to be whitish. In March she was said to have suddenly lost her sight when washing up the breakfast things, and she complained at the time of sudden shooting pains in the eye. In May her mother was confident that she could not see. The left fundus oculi was then normal. The right optic disc was of paper whiteness, and the arteries were very small and tortuous. She complained of continuous headache, and she blundered into the furniture as she walked. In November, 1889, she developed epilepsy. In May, 1890, her knee-jerks were exaggerated, and right ankle-clonus was marked. During voluntary movements the right arm became tremulous, but not otherwise: the hand was pronated and strongly adducted, and the triceps and supinator jerks were exaggerated. There was no facial paralysis. In June the arteries of the left optic disc were slightly tortuous, and the disc was pale: vision was defective, but she fixed with it. In March, 1891, she was still epileptic, and complained of headache and giddiness. In October, 1892, she was wet and dirty; she still had fits, and was becoming more demented, and she also had delusions. From then to December, 1895, her retrograde progress was slow but steady. She was then bedridden and wet and dirty, but she had not had any fits for four months. In March, 1896, she was much worse, the fits were troublesome, and she died soon afterwards. At the necropsy, lymph a third of an inch in thickness was found enveloping the whole of the brain. The whole of the left side of the brain in all its parts was smaller than the right. The convolutions were flattened. The ventricles contained but very little fluid. The vessels at the base were normal.

The Chairman (Dr. Henry Ashby) (Manchester) replied that in their paper they brought forward facts to show that a temporary amaurosis sometimes followed severe attacks of infantile convulsions, and they sought to distinguish this group from the better known group of cases of amaurosis which follows an attack of posterior basal meningitis. In the former or post-

eclamptic group they suggested that the blindness was due to a discharge from the visual centres, followed by a condition of anæsthesia. They need hardly say that they were conscious of the difficulties attending the diagnosis of the cause of the attacks of infantile convulsions in many instances, and also that meningitis or a local encephalitis might be easily overlooked during infancy, especially in its initial stages. But, putting doubtful cases on one side, and taking the typical cases to which they called attention and which came under their observation, they could not agree with Dr. Carpenter in thinking that these were in reality cases of meningitis. Infants commencing with meningitis might or might not have convulsions, but no one would pretend that meningitis is a common cause of convulsions: indeed, the whole history of eclampsia and epilepsy disproved this. Surely, because a neurotic infant has a feverish attack and severe convulsions ending in recovery, they were not justified on such symptoms alone in diagnosing meningitis, even though the attack might have been followed by a fleeting amaurosis. Certainly, as a rule, if meningitis commenced with convulsions the more characteristic symptoms of cerebral disease followed: the common sequence was not fever, convulsions, coma, and recovery. The pathology of the temporary blindness, hemiopia, aphasia, or paresis which sometimes follows convulsions or epileptic attacks, was far from certain: there might be punctiform hæmorrhages, toxin poisoning, or, as they suggested as a cause for the amaurosis, a condition of exhaustion following discharge. That the more lasting hemiplegias or other paralyses which were associated at times with convulsions or whooping-cough, either one or both, were due to meningeal hæmorrhage resulting from the stress and strain consequent on the convulsions and respiratory spasm, was, they thought, very probable, and it was quite possible that the temporary loss of function which took place was due to a similar cause. With regard to another point raised as to the condition of the pupils and their reaction to light, their own experience was that in the early stages of amaurosis following undoubted cases of post-basal meningitis in infants the pupils were dilated and sluggish, or did not react at all: later, as sight returned, the pupils reacted normally. This, however, did not seem to agree with the observations of some others. In their own cases of post-eclamptic amaurosis the pupils were active from the first.

45. A CASE OF PNEUMOCOCCUS ARTHRITIS IN AN INFANT.

By A. H. TUBBY, M.S.

Ernest F—, aged 14 weeks, was admitted into Westminster Hospital on March the 6th, 1903, on account of pain and swelling about the right knee. The history was that ten days before the child, who was previously healthy, became fretful and cried when he was moved, especially when the right leg was touched. His mother stated that he had been treated for rheumatism. Eight days earlier the right knee became red, hot and swollen, and the child was very feverish. No history of any injury could be obtained, but the mother stated that of her three children one had just died from diphtheria, while the other was then under treatment at a fever hospital.

On admission the child was evidently very ill, and the temperature was 102° F. The right knee was globular in shape, very red, and the swelling extended a considerable distance up the thigh and down the leg. The knee was flexed, and any attempt to move it caused extreme pain. It was freely fluctuating, but the fluctuation was limited to the capsule of the joint, and no pus appeared to have escaped into the periarticular tissues. The child was anæsthetised and the joint freely opened by two lateral incisions. About an ounce of pus escaped. The joint was freely irrigated with sterilised water, and a drainage-tube was passed across it. The pus was examined by Dr. Lazarus Barlow, Pathologist of Westminster Hospital. He reported as follows. "Cultivations from the pus show a streptococcus form of what appears to be a diplococcus. Without animal experiment it is impossible to speak with certainty. But the appearances are not inconsistent with the view that it is *Micrococcus pneumoniæ*. Indeed, the naked-eye aspect of the culture in the tube is typical of that organism. A diplococcus in fair numbers is present in the pus itself in the joint."

The child lay in a perilous condition for several days, and the temperature did not subside until the fourteenth day after admission, or the twenty-fourth after the illness. By that time the drainage-tube had been removed and the wounds were granulating healthily. Frequent examinations of the chest were made, but no pneumonic signs were discoverable. The joint is now partially ankylosed, which is likely to become complete.

Remarks.—This case is of interest because of the attention that has been directed to this subject. In an article by Dr. Nathan Raw in the 'British Medical Journal' of December, 1901, several cases are quoted; six of these were suppurative and one serous. Of these, three died and four recovered. He alludes to the work of Lerous on the subject, who up to 1899 collected twenty-eight cases, and Cave of Bath has recorded three other cases. The writer had seen two cases during the past year at the Evelina Hospital, which were verified by microscopic examination and animal inoculation. In one of these cases, an arthritis of the hip, pneumonia pulmonis preceded the joint trouble; and in the other, an arthritis of the knee, there was no lung complication. The discovery of a pneumococcus arthritis undoubtedly threw considerable light upon the pathology of those cases of acute arthritis of infants described many years ago by Sir Thos. Smith. (*Read March the 20th*, 1903.)

Discussion.

Mr. Lockhart Mummery asked whether Mr. Tubby had tried dividing the patella in such cases when occurring in infants. It seemed a very sound principle, because it was difficult otherwise to get adequate room for doing anything in the joint, or even to insert a drainage-tube of any size. Division of the patella, which was easily done by the knife in children, had been recently written upon. Another point was that the child should be nursed in such a posture that the knee-joint became the lowest part, to facilitate the escape of pus. A splint should not be used.

Mr. A. H. Tubby, in reply, said that he had not divided the patella in children, because he did not wish to have much surface exposed. With regard to drainage, he preferred to use two tubes, one on either side, and not to place one right across,

because the less the amount of granulation set up by the presence of the tubes the better, and the less the likelihood of ankylosis occurring afterwards.

46. A CASE OF ICHTHYOSIS HYSTRIX (ICHTHYOSIS HERPETIFORMIS OF HUTCHINSON).

By DAVID WALSH, M.D.

This is a well-marked instance of an uncommon congenital affection of the skin, especially rare on the neck and face. The condition was noticed shortly after birth, and within the last two years the portion on the side of the neck has become verrucose and deeply pigmented. The part of the affected skin stretching over the lower jaw shows a hypertrophic condition somewhat like cheloid, but without warts or pigmentation, and is sharply limited at the middle line. A discoloured linear macule stretches away backwards towards the lobe of the right ear. The child is 6½ years of age, and its previous health was good, except convulsions when teething. There are two other children, who are healthy, and none show any affection of the skin. The family history is good.

This congenital condition is usually asymmetrical. It often occurs in lines along a limb, but does not correspond closely, as a rule, with nerve distribution. The probable explanation appears to be that it is a developmental defect, possibly influenced by atavism. The later development of warts and pigmentation in these ichthyotic patches is not uncommon. The treatment consists in snipping away the warty growths with a curved scissors.

(*Exhibited April the* 17*th*, 1903.)

Discussion.

Mr. George Pernet said he did not consider the name ichthyosis hystrix was applicable to Dr. Walsh's interesting case. He differed from the exhibitor in two points, viz.: it did not suggest a keloid, and it did *come* into the category of warty streaks. Indeed, the greater part of the growth was of the nature of a white mole. As to etiology, it was developmental.

That was as far as we could go. As to Dr. Walsh's view that it was atavistic, the speaker asked if by that the exhibitor meant that the condition could be traced back to our shark-like ancestors! Owing, perhaps, chiefly to Lombroso, the word atavism had come to be used in rather a loose manner.

Dr. David Walsh, in reply, said there was considerable difficulty in attaching a name to a condition of this rare kind, but on the whole it appeared to approach ichthyosis hystrix more nearly, perhaps, than any other named affection. The course of development of the wall of the face was from behind forwards to the anterior mesial line, where it joined the opposite half of the face. Imagine a defect beginning in front of the ear, and it would conceivably follow very much the course shown in this patient's neck and face. It was as if a stone were placed in the middle of a stream advancing with a strong, slow curving current, and finally stopped dead by a transverse barrier. The suggestion that this condition might be called a "white mole" did not further matters. Whatever the growth might be, it was neither white nor was it a mole.

47. A CASE OF DEXTROCARDIA.

By PORTER PARKINSON, M.D.

Dr. George Carpenter showed for Dr. Porter Parkinson a boy 6 years of age, who was admitted into the North-Eastern Hospital for Children on April the 12th, 1903, with an attack of rheumatism. When first seen he was a well-nourished child, with a rheumatic effusion into the knees and ankles. The heart's apex-beat was not defined, but there was marked epigastric pulsation. The heart-sounds were best heard on the right side, one inch below and half an inch internal to the nipple, and the area of cardiac dulness was on the right side. There was a faint systolic murmur over this area. The liver was in the normal position. Under salicylate of sodium the pain and effusion into the joints quickly disappeared. (*Exhibited April the 17th*, 1903.)

Discussion.

Dr. Edmund Cautley thought there was no doubt about it being a case of dextrocardia. He agreed with what had been said, except that the apex could be both felt and seen about half an inch to the inner side of the nipple on the right side.

48. A CASE OF SCURVY IN A CRANIOTABIC INFANT, WITH OBSERVATIONS ON CRANIOTABES.

By GEORGE CARPENTER, M.D.

Lottie S—, aged 9 months, was brought to the North-Eastern Hospital for Children for hæmaturia in April, 1903. She had been fed with malted food and Nestlé's milk made with boiling water,—nothing else.

On examination it was found that she had cut the two upper and two lower central incisor teeth. Around the upper incisors, and also over the gums adjacent, corresponding to the two lateral incisors, where there were apparently no teeth, the gum was violet-coloured and very spongy and superficially necrotic. The lower gum was unaffected. The mother said she did not use her legs, and she resented interference, but no thickening was detected. There was marked beading of the ribs, and the wrists were a little enlarged. She was *anæmic*, but there were no cutaneous hæmorrhages. Craniotabes was marked over the parietal bones, and was found also on the occipital and temporal bones. Loud "snuffles" had been noticed from birth, but there was no discharge *viâ* the anterior nares. She was a twin and the only child; the other was born dead. There were no miscarriages. She had "thrush," but this did not go below the buttocks.

She was ordered the juice of one orange daily, and a suitable dietary prescribed. The urine was examined and blood was found in it, but there were no casts and no oxalates.

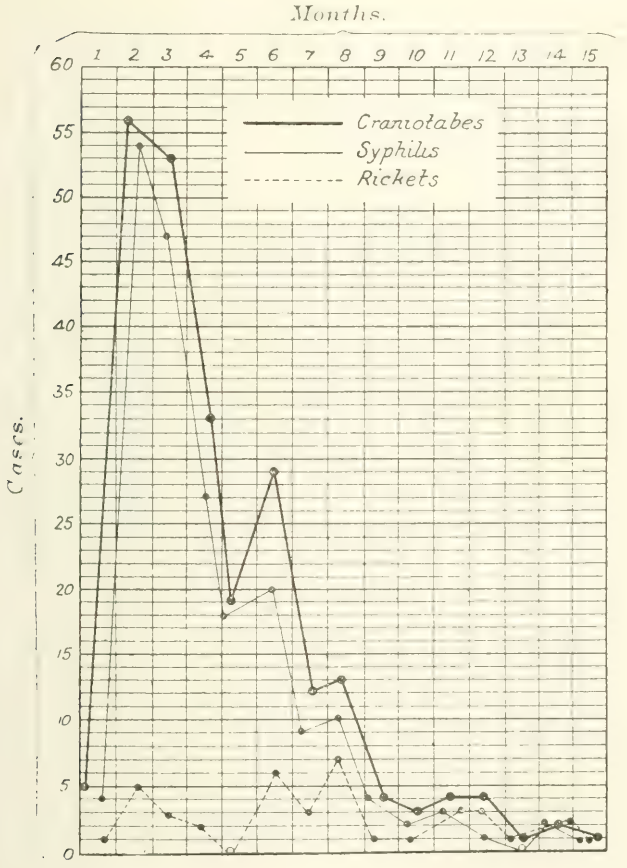
On April the 10th the child was much better. The gums were much less swollen, and there was then but a faint trace of purple discoloration to be seen. The lateral incisor teeth were then just visible; these could not be seen on the last visit, owing to the swollen condition of the gum.

The *urine* contained a trace of albumen, and seemed to

be free from blood, but there was a *trace* with guaiacol and ozonic ether. She was very anæmic.

On reference to the text-books one found it still stated that craniotabes was most commonly found in the occipital

Fig. 21.



bone, and not in the parietal bone, or only occasionally in the latter. That was not his experience, and he had years ago drawn attention to the fact that craniotabes was usually to be found in the parietal bones, and least likely in the

frontal bone, and then only when the other skull bones were attacked. As to the interpretation of craniotabes, his impression was that more often than not it was a syphilitic manifestation. Some years ago he collected a large number of cases (238) of craniotabes. In a chart which he had prepared (*vide* Fig. 21, page 215) craniotabes was represented by a very dark line, syphilis by a thin unbroken line, and rickets by a broken line. It was seen that craniotabes was most common during the second, third, and fourth months of life, and was very infrequent indeed at about the ninth month. Thus, during the pre-rickety period craniotabes was a marked feature; and when rickets was marked craniotabes was found with difficulty. He found it somewhat difficult to make up his mind as to the meaning of chronic "snuffles," whether it was a manifestation of syphilis or not. In a certain proportion of the cases adenoids were to be found in the posterior nares, while in others there were none. Some had a muco-purulent discharge from the anterior nares; others had not, but in some cases the muco-pus passed by the posterior nares, and could be seen at the back of the pharyngeal wall. He thought it would be interesting to hear an expression of opinion as to the value of *chronic* "snuffles" in infants. (*Exhibited April the 17th*, 1903.)

Discussion.

Dr. Edmund Cautley thought the case certainly raised many interesting points, apart from the condition for which it was brought. One should not lay too much stress upon "snuffles." A very large proportion of babies seen in hospital had "snuffles," or at least some kind of nasal catarrh. But in private practice more stress might be laid upon it. He could not agree with Dr. Carpenter that craniotabes was very frequently associated with syphilis, and asked that gentleman whether, in compiling his statistics, he had diagnosed syphilis from the "snuffles" alone, or were there other signs of the disease present? With regard to the situation and varieties of craniotabes, he thought there were three distinct types. There were cases, very rare, in which practically the whole vault of the skull was membranous. Those were cases of congenital defect, and were usually associated with congenital hydrocephalus. There

were also cases described as typically syphilitic, and mentioned in Dr. Carpenter's book, where there were isolated areas of craniotabes, especially marked in the parietal bone. Those cases were so uncommon that he had seen very few of them. The more frequent type was analogous to the present case, in which there was softening in and about the occipito-parietal suture ; and it always seemed to him that the softening was due either to delayed ossification of the edges of the parietal and occipital bones, or possibly to the reabsorption of the salts already deposited there. He agreed that those cases were most commonly seen at about the fourth month, or from the second to the fourth ; and he believed they were much more frequently seen in the early months of the year. He had gone through a large number of cases in two years, and found that nearly all the babies who suffered from craniotabes at that period were fed otherwise than by the breast, viz., on condensed milk or food which had been boiled. It was a matter of experience that if milk was boiled or cooked in any way the salts in it were rendered less soluble, and it seemed reasonable that craniotabes of that nature should be due to a deficiency of salts in the food. Therefore he thought craniotabes of that type was not at all associated with syphilis, but was due entirely to errors in the mode of feeding, which later on might lead to rickets or other disorders of nutrition. In connection with syphilis, one might say syphilis predisposed to it to the extent that a syphilitic child might be debilitated and its nutrition more impaired. In the form pictured in Dr. Carpenter's book he thought it might possibly be associated with, and dependent upon, congenital syphilis, but that beyond this craniotabes was not due to congenital syphilis, but to errors in diet. The present case also showed the condition called venous dystrophy, venous dilatation on the scalp, which a French author sought to connect with syphilis. He (Dr. Cautley) thought it was more likely to be due to the general malnutrition with consequent thinning of the skin.

Mr. Sydney Stephenson said he believed that Dr. Carpenter, in describing the case, spoke of the upper incisors as being the only teeth erupted, and yet referred to the gum changes as being general. He (Mr. Stephenson) was under the impression that the gum changes in scurvy rickets only showed themselves at spots where the teeth had come through.

Dr. G. A. Sutherland said the subject of craniotabes was a very difficult one, and he believed it had not yet been settled whether it was due to rickets or to syphilis. He agreed with Dr. Cautley that it was a condition found but rarely ; it certainly was not nearly so common as the text-books would lead one to believe. He was inclined to believe it was syphilitic in origin. In rickets he did not think there was the typical

craniotabes which had been referred to, where there were simply larger or smaller spaces in the bone which were felt to be membranous. The parchment-like areas in the skull which could be depressed were often seen in rickety skulls, but that was not the typical craniotabes condition, in which there was simply a definite disappearance of bone in small areas. He had post-mortem specimens showing it, the areas varying from the size of a pin's head to that of a shilling, scattered over the parietal, occipital, and frontal regions. In his experience the parietal bone was the most frequently affected, but in some cases it was singular how generalised the condition was in the skull bones. Where the hollows were only small the condition could not be detected during life. "Snuffles" was another difficult problem, and it was hard to say whether a given case was specific or non-specific. "Snuffles" was not to be regarded as alone diagnostic of syphilis. In most of the cases of syphilis other symptoms of the disease could be seen. The condition with which "snuffles" was apt to be confused was adenoids. Some medical men, whose opinions he respected, found adenoids to be very common in early infancy, but his own experience was that they were very rare. If there was any doubt during the first two or three months of life as to whether a case was due to adenoids or syphilis, he would say syphilis was very much more likely than adenoids. It was surprising how many children were brought because of hæmaturia, especially infants under the age of twelve months. During the past year there had been quite a succession of such cases at the Paddington-Green Hospital, all of them associated with scurvy. At the autopsy on one case the pathologist found in the bladder what he believed to be a nævus. On further examination, however, it was found to be extravasation of blood under the mucous membrane of the bladder, and this suggested that vesical as well as renal hæmorrhage might occur in scurvy in infants.

Mr. John Stuart Mackintosh (Hampstead) said he understood "snuffles" was usually regarded as a muco-purulent discharge in the nose in a young baby, producing a snuffling noise. That noise might surely be produced by conditions other than syphilis and adenoids, and he believed it was often caused by coryza. Again, during the child's inspiratory efforts at parturition, it could draw into the upper respiratory tract liquor amnii, or a purulent discharge in the vagina, which might cause a rhinitis passing into a subacute or chronic condition. He had seen many young babies with congenital adenoids, or where they had developed during the first few weeks of life, and that condition had been spoken of by the mothers as "snuffles." Only the other day he cleared out the naso-pharynx of a baby, and the snuffling noise ceased. Usually they were difficult to demonstrate in such young children, but that could be done by

having in one hand a pair of oral forceps, and picking up the adenoids at the back of the pharynx at the moment of removal.

The Chairman (Dr. Lewis Marshall) (Nottingham) believed that "snuffles" was talked about in the loosest possible way in connection with congenital syphilis. In his experience children suffered from "snuffles" from causes which were unimportant; it would be very rash to arrive hastily at the conclusion that children who "snuffled" were syphilitic necessarily. He agreed with Dr. Cautley and Dr. Sutherland that craniotabes was not as common as generally supposed, and when it existed he did not attribute it to syphilis. He thought there were general causes, such as malnutrition, which might induce such a condition. Syphilis might be a modifying influence. He did not think Dr. Carpenter had very clearly made out that his case was one of scurvy rickets; he did not notice mention of subperiosteal hæmorrhages or purpuric eruption. Hæmaturia was very common in children, and the causes were often quite unimportant. One of the commonest was lithiasis. It was only too common in children fed upon malted food and Nestlé's milk. Hæmaturia, when contrasted with that symptom in adult life, was not necessarily a grave symptom in children. With regard to scurvy rickets generally, his own feeling was that the cause of it was an imperfect milk supply, pure and simple. He had recently seen, in the midland counties, three or four well-marked cases of scurvy rickets which were the direct products of Mellin's food. They improved immensely by proper feeding—that spoken of by Dr. Ashby, of Manchester, as feeding by prescription, *i. e.* adjusting the fats and casein of the milk.

Mr. Henry Skelding (Bedford) asked what medicinal treatment the child had before it came to the hospital. Some years back he saw a child which was thought to have scurvy by reason of spongy gums, and it was found that before admission into hospital it had been having Hydrargyrum cum Cretâ.

Mr. George Pernet said that with regard to "snuffles" he would not diagnose congenital syphilis on that condition alone, but that true "snuffles" was of syphilitic origin he had no doubt. As to craniotabes, he had examined the skulls of congenital syphilitic infants for this condition. It was not common. As far as he was concerned, he considered it very suggestive of syphilis, and a factor to be taken into account with others. He quite agreed with Dr. Cautley that it might occur associated with rickets (malnutrition); but his experience in that connection was small as compared with that of those connected with medical departments at children's hospitals. Nor did he wish to be considered as an authority on scurvy rickets. But he had noticed in the infant shown by Dr. Carpenter how pale it was, owing to the improper feeding with material deficient in iron.

Von Bunge, of Basle, in his interesting 'Physiologie des Menschen,'* had called attention to the danger of an insufficiency of iron in the food of children. Acting on Von Bunge's work, Heubner, of Berlin, had advised the early daily use of thoroughly cooked green vegetables (a teaspoonful of spinach, etc.) to a child that had cut, say, eight teeth.

Dr. George Carpenter, in reply, said the spongy gums were around the upper incisor teeth, not the lower. His first impression was that the upper lateral incisors had not been cut, but that was found to be incorrect when the swelling subsided somewhat. Occasionally the gums became spongy at places where there was no sign of erupted teeth. No doubt hæmaturia did occur in children who were admittedly suffering from scurvy, *i. e.*, children who had subperiosteal hæmorrhages. The present child had not those hæmorrhages, but the scurvy was unmistakable, because of the great improvement brought about by change of food. However, subperiosteal hæmorrhages and cutaneous hæmorrhages were not essential to the scorbutic condition; nor was rickets, the term scurvy rickets being a misnomer. Scorbutic children were by no means always rickety, any more than they were necessarily anæmic, and mucous membranes of good colour were not uncommon. He had never seen sponginess of gums produced by the administration of Hydrargyrum cum Cretâ, as mentioned by Mr. Skelding, and he had given large quantities of that drug for years past. He (Dr. Carpenter) suggested that the Hydrargyrum cum Cretâ might have been given for syphilitic reasons, and that scurvy was simply a complication. Mercury, in small doses, increased the number of red blood-corpuscles. In children, both old and young, hæmaturia indicated scurvy very often. Children were occasionally admitted into the wards who were suffering from hæmaturia only. They apparently had no stone, and under a proper hospital dietary the hæmaturia disappeared. He believed those were cases of scurvy. Mr. D'Arcy Power showed before the Society about a year ago a case of hæmaturia supposed to be due to a stone in the kidney. Operation, however, revealed no stone; but the child subsequently developed typical scorbutic gums, and recovered under anti-scorbutic treatment. He had brought the case before the Society because it illustrated what he took to be an important point which was not generally recognised, viz. the fact that hæmaturia in infants and children usually denoted scurvy, and, moreover, was not infrequently the *only* sign of that disorder. He admitted there were great difficulties in the way of deciding about the nature of craniotabes, but in a certain number of obviously syphilitic children craniotabes occurred. Other cases had come before him in

* 1901, vol. ii, p. 493.

which he could find nothing but "snuffles" and craniotabes, with or without splenomegaly. In such cases he found brothers and sisters who had typical syphilitic eruptions, or other evidences of that disorder. He had also had other cases where he knew one or other of the parents to be syphilitic, and where the only indication in the child was craniotabes and "snuffles." He had found in some instances one infant of the family markedly syphilitic, and the infant next born craniotabic and without obvious marks of syphilis. Craniotabes, in his experience, was not at all uncommon, and when speaking of craniotabes he meant definite atrophic patches in the vertex, which could be diagnosed by the usual clinical methods, and he did not confuse this condition with that of some thinning along the suture lines; on the contrary, he was careful to exclude such cases from his statistics. He had paid special attention to craniotabes, and looked for it as a matter of routine, and that was perhaps why he found it more frequently than others who were, perhaps, not so specially interested in the condition; but he had given his views and experiences on the subject, and there was no doubt in his mind—so much so that when he found craniotabes he always *suspected* syphilis. When speaking of "snuffles" he, of course, referred to a chronic condition, not to a simple cold in the head, which would soon pass off. In a certain number of cases of "snuffles" one could digitally examine the naso-pharynx, and there were no adenoids. In others they were present, and in others the finger could not be got to the posterior nares by reason of the smallness of the parts. Whether some of those cases were syphilitic or not was a difficult question, and he often felt puzzled when chronic "snuffles" was the sole symptom.

Postscript (August the 20th, 1903).—The child is now 13 months old. The craniotabes has disappeared, the skull being quite hard, and has given place to a typical hot-cross-bun skull (Parrot's nodes); the anterior fontanelle is patent. She has a muco-purulent discharge from the nose; there are no adenoids; the nostrils are very small. The mucous membranes are a little pale. The upper central incisors are carious, and one of the lateral; she is cutting the first premolars; she has all the incisors in the lower jaw. The ribs are decidedly beaded, there being a rickety rosary, and the wrists are perhaps a trifle enlarged. She has a pot-belly. The urine is now normal. Since the recovery from scurvy she has been treated by a teaspoonful of steel wine and three minims of

Fowler's arsenical solution thrice daily. Mercury has not been administered. I should like to draw special attention to the disappearance of the craniotabes, although the child is still rickety, and to its replacement by Parrot's nodes, which I deem to be a syphilitic manifestation.*

49. DEFORMITY OF THE HANDS AND FEET TRANSMITTED THROUGH FIVE GENERATIONS.

By LEWIS MARSHALL, M.D.

I am induced to bring the history of this family before the Society because the transmission of any deformity is uncommon in my experience and I wish to elicit from members present their experience, on this point.

R. T—, a baby (female) 7 months old, was brought to me in the early part of this year for treatment of some simple infantile disorder. I then noticed that the child's hands were deformed, each finger stopping short at the proximal phalanx, and the thumbs were ill-developed. The mother told me that the child's feet showed a less evident defect, and on examination I found that the two outer toes on either foot were small and ill-proportioned. The mother's hands and feet I afterwards discovered were exactly similar to her baby's. In this family there was one other child—a girl—whom I saw, and she suffered in the same way. At this interview the fact came out that the maternal grandmother, who was still living in this city, had hands and feet like those already described. By calling on this old woman I added the following facts to those I have given, viz. that her mother (*i. e.* the great-grandmother of my patient on the maternal side) had borne twelve children, and of these alternate children had the defect which I had seen in three generations. She was unable to

* Of my cases, 57·5 per cent. were demonstrably syphilitic, *vide* 'Syphilis in Children,' page 73.

tell me in what degree the sexes were affected. The mother of these twelve children had no deformity, but her husband "had all his fingers shortened, and double thumbs." In the next, the fifth generation, the mother of the man last described was deformed exactly in the same way. The children in this family were affected, but only alternate children, and here again I was unable to glean what was the sex of the deformed children. A collateral branch in the fourth generation showed the defect which had been transmitted by a female to her son and by him to

Fig. 22.—Hands of patient's maternal grandmother.

his only child, who was a male. The last two members mentioned in this line are living in Nottingham.

In those cases examined by me the stumps were all like those which are seen in the so-called intra-uterine amputations of legs or arms, and of which I have seen a few instances. The facts reveal that in the fourth and fifth generations some children suffered whilst others escaped.

The regularity with which this difference in the offspring appeared is remarkable, as is also the faithful manner in which the deformity was reproduced. Such a persistent

repetition can only be explained by heredity. The theory of Hasse and Pauli as to the production of such deficiencies by an early adhesion of the amnion and integuments cannot apply to these cases. Mr. Hutchinson, in his 'Pedigree of Diseases,' p. 27, when speaking upon the idiosyncrasy of structure, touches upon the "freaks of nature, such as moles and nævi and the like." He goes on to say, "In the idiosyncrasies, however marvellous some of them may be, there is usually to be noticed a

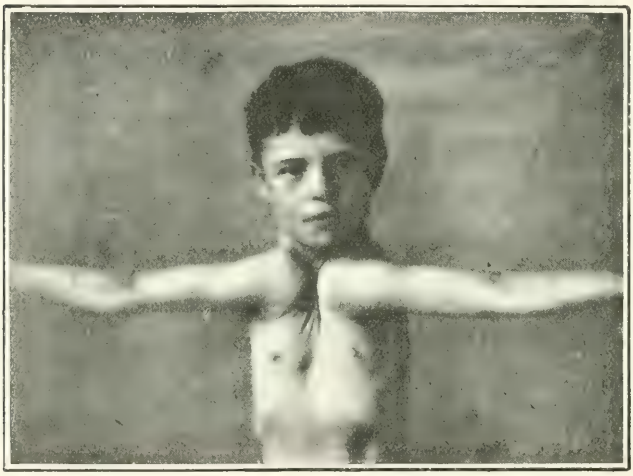


Fig. 23.—Dr. George Carpenter's case of congenital absence of the clavicles in a girl 8 years old.

certain reference to law and order. They do not occur absolutely by chance, and they often appear to be correlated with other less startling peculiarities, and they often happen to several members of the same family, or are distinctly hereditary." I have quoted this passage from Mr. Hutchinson's lecture so freely because the history which I have given is strictly in accord with the statements there made by him. The only similar case to my own that I am able to find a record of is given in Keating's 'Encyclopædia of Children's Diseases,' by Carson of Cincinnati.

Dr. George Carpenter has published * a case of absence of the clavicles with an account of various deformities of the clavicles in five other members of the same family, the accompanying photograph illustrating the condition, and I am indebted to him for permission to reproduce it. Dr. Alfred Lingard has also drawn attention to hereditary transmission of hypospadias, and its transmission by indirect atavism. Dr. Lingard also gives a long list of references bearing upon the question of transmission by heredity.† (*Read April the* 17*th*, 1903.)

Discussion.

Mr. George Pernet wished to know if Dr. Marshall had inquired into the condition of the descendants of those children who had escaped the hereditary action, and had been born with normal extremities.

50. A CASE OF COLLOID CANCER INVOLVING THE PERITONEUM AND LARGE INTESTINE IN A GIRL AGED TWELVE YEARS.

By J. PORTER PARKINSON, M.D.

The infrequency of this disease at an early age, and certain points of interest in connection with the origin of the growth and its special symptoms, appear to be a sufficient reason for bringing it before the notice of the Society.

Frances D—, aged 12 years, was admitted into the London Temperance Hospital under my care on November the 7th, 1902, for swelling of the abdomen. There was a history of phthisis on the father's side, but no other points of importance. There was no family history of cancer. The patient had had all the usual infantile illnesses, and also typhoid fever. She was stated to have been always

* 'Lancet,' January the 7th, 1899.

† 'Ibid.,' April the 19th, 1884.

delicate, but for the past fortnight she had suffered from cough and shortness of breath on exertion, and the abdomen had only been noticed to be large for a week before admission, during which time there had been swelling of the feet, frequent micturition, and looseness of the bowels. The appetite was good, and there had been no vomiting.

On admission she was wasted and anæmic. The temperature was normal. The tongue was clean. The abdomen was greatly distended (girth 32½ inches) with free fluid; it was uniform, with bulging in the flanks; the area of resonance above was small in area and the note somewhat impaired. There was some œdema of the wall of the abdomen. No organs could be felt. She passed about five or six loose, offensive motions daily. The heart's apex-beat was in the nipple line, in the fourth intercostal space, and there was a systolic præcordial murmur, probably hæmic.

There was some loss of resonance at the bases of the lungs posteriorly, but otherwise there were no abnormal physical signs in the chest. The urine was diminished in amount, being of specific gravity 1024, acid, and at times containing a faint trace of albumen. The patient was thought to be suffering from tuberculous affection of the peritoneum with effusion, and on account of the pressure symptoms the abdomen was tapped on November the 13th, and eight pints of fluid withdrawn. This fluid was slightly alkaline, of specific gravity 1012, boiled solid with albumen, and on microscopical examination showed a few leucocytes and no corpuscles, and also some larger cells containing spaces, drops and granules of a transparent material resembling fat, but not showing the high refraction of the latter. These cells were filled with what was evidently a colloid deposit (*vide* Fig. 24, page 227). On examining the abdomen after tapping, an irregular, hard swelling could be felt in the hepatic area, and some nodules could be detected evidently free from this. The spleen was just palpable.

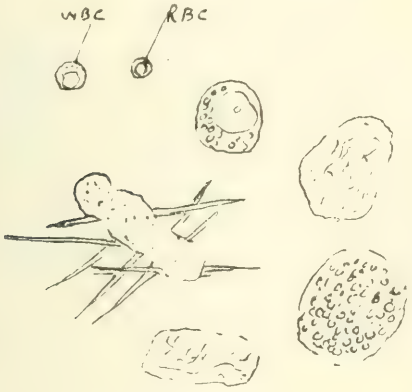
On December the 2nd an exploratory laparotomy was performed by Sir W. J. Collins, who found the peritoneum

sown over with small masses of growth of a sodden, semi-translucent appearance, and varying in size from a pin's head to a pea, or even larger. A nodule was removed for examination, and proved to be colloid cancer. The wound healed by first intention.

From this time till her death on February the 28th there is nothing to record except steady wasting and gradual increase in size of the lumps in the abdomen, while the fluid did not reaccumulate to any extent. Eventually the abdomen appeared completely filled with the irregular masses of growth.

At the autopsy, on opening the abdomen the thickened

Fig. 24.



omentum had to be peeled away from the abdominal wall before the abdominal cavity was exposed; four or five pints of fluid escaped. The peritoneum was seen to be covered with coalescing masses of colloid material, which involved all the organs, but especially the great omentum, which was much thickened, and the whole of the large intestine, the wall of which was infiltrated, without, however, any great diminution of calibre. The small intestines were less affected. The various organs were matted together by the growth, and the liver embedded in a mass of colloid material of about a quarter of an inch in thickness. The

spleen was similarly affected, and the stomach was less involved than the other viscera in the deposit. The photograph shows the large amount of cancerous growth.

The primary seat of the growth appears, both from its microscopical structure and the gross appearances, to be in the rectum.

There are two points in the clinical aspect of this case which I should like to mention; the first is that there was considerable enlargement of the superficial veins over the

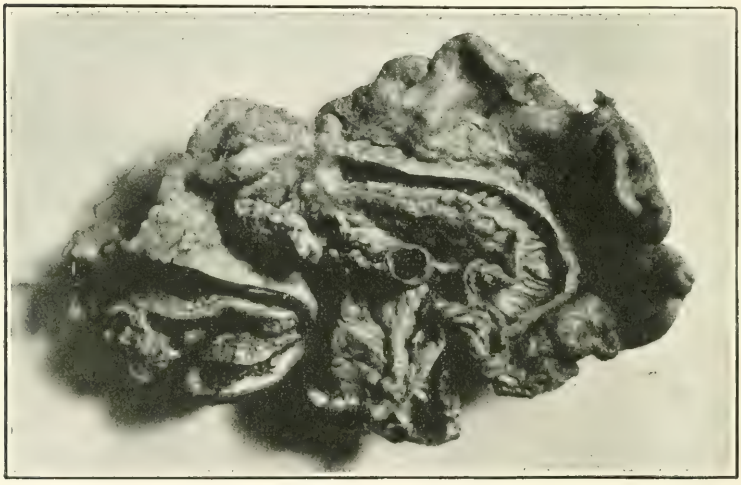


Fig. 25.—Colloid cancer of peritoneum. *a*, rectum; *b*, duodenum; *c*, colon.

upper zone of the abdomen, similar to what is met with in cases of cirrhosis of the liver; this was obviously due to interference with the portal vein or its tributaries by the masses of growth. I may incidentally mention that the same venous enlargement is not uncommonly seen in cases of tuberculous peritonitis.

The second clinical point is, that the nature of the cells in the peritonitic fluid removed by the first tapping might have been recognised as cancer cells if the likelihood of

that disease had not been overlooked, owing to the youth of the patient.

In the cases of colloid cancer of the peritoneum I have seen or read of, the amount of fluid in the abdominal cavity was insignificant in amount, but in this case it was the first symptom, and was for two months the most marked feature.

The growth consists of a reticular, nucleated, fibrous tissue, arranged in bands or strands, in the meshes of which are cells of various sizes and shapes, which for the most part have undergone a colloid change, but which here and there can be distinctly seen; in one or two places these cells are typically columnar, and line alveoli like those of an ordinary columnar epithelioma.

The youngest case of this disease of which I can find an account was recorded by Drs. Kanthack and Furnivall in 1896 in the 'Transactions of the Pathological Society.' It was in a boy aged 19 years, and the primary growth involved the cæcum and ascending colon and the lower two or three inches of the small intestine, and caused symptoms of intestinal obstruction for which colotomy was performed, the obstruction of the lumen of the bowel being nearly complete. The secondary deposits were much less numerous and extensive than in the case now reported.

(*Exhibited May the 15th*, 1903.)

Discussion.

Dr. Theodore Fisher (Bristol) wished to ask Dr. Parkinson whether he had found where the growth primarily originated. Although the great majority over the peritoneum were secondary to disease somewhere in the alimentary canal, it was sometimes difficult to find the primary source. He had been unable to do so in one case in the post-mortem room, and yet the sections had shown the growth to be carcinoma, as in the present case. Dr. Parkinson's case showed a very common appearance of carcinoma in the stomach and also the intestines, viz. the very great hypertrophy occurring in the coats of the stomach. One could sometimes see that in the intestines also, but it was not so well marked there as in the stomach in the case under discussion. The muscular hypertrophy frequently occurred without any evidence of obstruction of the lumen of the stomach or intestines.

Dr. A. E. Sansom thought the case was a very interesting one and probably a record, as the patient was only twelve years old. He had nothing to contribute to the discussion, but from the practical point of view he would ask Dr. Parkinson whether an examination of the fluid might possibly throw light on future cases in the way that he could put down certain things as colloid.

Dr. J. Porter Parkinson, in reply to Dr. Fisher, said that in looking at the growth near the rectum one saw it extended pretty well through the whole thickness of the gut. It involved almost the whole of the rectum; certainly for some distance up it involved the mucous membrane of the rectum, and in that part the muscular coat could be seen. It could be seen in the microscopic sections which he would like to have shown. As to whether the fact of its being epithelioma meant that it sprang from the bowel he did not know. Dr. Kanthack, in a paper read some time ago before the Pathological Society, seemed to think that the finding of very definite columnar cells suggested that it proceeded from the alimentary canal. With reference to Dr. Sansom's remark about finding the cells in the fluid, he thought the drawing which he had passed round showed the large size of those cells which were to be found there compared with the blood-corpuscle which was also there. They were exceedingly large, and there were many of them which were obviously filled with translucent material, and were unlike any other cells he had seen. He was not certain whether a future case could be recognised by their presence. He thought it might point to the fact of the case being possibly one of cancer if it occurred in another patient.

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51. UNUSUAL JOINT CHANGES IN A CHILD THE SUBJECT OF INHERITED SYPHILIS.

By A. A. H. PARTRIDGE, M.D.

(Introduced.)

Albert H—, aged 10½ years, developed interstitial keratitis in the left eye in November, 1901, and the right eye became affected in January, 1903. Soon after the left eye was attacked the finger-joints of both hands became swollen. The left hand was affected before the right. At

present the first phalangeal joints of all the fingers are enlarged, and also the metacarpo-phalangeal joints of the right hand. The enlargement seems due to the tissues round the joint rather than to enlargement of the bones, and is very like the condition found in rheumatoid arthritis. No other joints are affected. The head has the appearance of congenital syphilis. There are scars at the corners of the mouth. The teeth are pegged, but the central incisors are not notched. The eyes have always been prominent, and the boy has been growing rapidly since the condition began. There is slight deafness of both ears. There is slight lymphatic enlargement in the neck.

(*Exhibited May the* 15*th*, 1903.)

Discussion.

Mr. Sydney Stephenson said it would be interesting to know if the toes were also affected.

Dr. Edmund Cautley said the case was very interesting on account of the association. It did not seem to him to be a syphilitic affection of the joints: he was much more inclined to think it was a case of osteo-arthritis occurring in a child the subject of congenital syphilis. The condition of the joints was so comparable to what was found in ordinary osteo-arthritis in children that he saw no reason to suppose it was syphilis in itself.

Dr. G. A. Sutherland asked if the patient had been treated for syphilis; if so, for how long, and whether any change in the joints had occurred under treatment. In connection with syphilis, one did have definite joint lesions occurring, and although if one were to take the fingers alone their appearance was very like that of osteo-arthritis, still, having syphilis in the case, and knowing that it had many manifestations, and would simulate almost any known form of disease, he thought those joint changes were probably syphilitic. One important test in that connection, of course, was the result of treatment. At the same time it was a very striking fact that in some of those syphilitic manifestations one did not get very direct benefit from treatment. With regard to the condition of the eyes, he believed most men were agreed that mercurial or other treatment did not have much influence in interstitial keratitis. In connection with joint changes they were often extremely slow to alter under any treatment, however carefully it might be carried out.

Dr. George Carpenter said that in some respects he agreed with Dr. Cautley that the condition was strongly suggestive of osteo-arthritis. There was no doubt about the child being syphilitic; the interstitial keratitis and the radiating scars about the mouth were very characteristic; he could not detect any splenic enlargement. He thought there was periosteal thickening of the heads of the metacarpal bones, and that the condition of the knuckles could not be distinguished from syphilitic epiphysitis. The sole point of distinction in the infant between rickety and syphilitic enlargement of the bone ends was the extension of periosteal thickening up the shaft of the bone. Under treatment that extension was the first to disappear, and then the joint was similar in appearance to a rickety enlargement. When periosteal thickening did not occur and there was no pseudo-paralysis, clinically the local conditions were indistinguishable. He thought that this was just such a case occurring in a child past the rickety age, and although there was no periosteal thickening extending up the shaft of the bone, the condition nevertheless, he thought, originated in the epiphysis. Syphilitic epiphysitis was comparatively common in infancy, but the age of this child did not exempt it. He did not think the thickening was altogether periosteal, and that one or two of the metacarpo-phalangeal joints displayed gummatous synovitis, though the majority of the finger-joints were unaffected. One point had escaped attention, and that was that the carpus on both sides was also involved and was decidedly thickened, which made its resemblance to osteo-arthritis the more striking. His own impression, however, was that it was a syphilitic manifestation, not one of osteo-arthritis, and some X-ray photographs should be taken of the joints, by which further information might be obtained. The condition was quite unlike that of syphilitic dactylitis, in which the shafts of the bones bore the brunt.

Dr. F. Parkes Weber said he had just examined the case very carefully with Dr. Carpenter, and the evidence was very strong in favour of the affection being due to congenital syphilis. There was complete absence of tenderness in the hands: the movement in the joints was almost perfect. One could move the fingers freely and the patient could move them freely himself without the slightest pain. The changes which one felt in examining the fingers seemed to be almost entirely confined to the bones and periosteum. Certainly there was just the slightest effusion into one or two of the joints, but when the periosteum in the neighbourhood of a joint was affected it would be strange not occasionally to get some effusion into the joint itself. Dr. Weber regarded the case as one of phalangitis of congenital syphilis. That affection, which had also been termed syphilitic dactylitis, had been very carefully studied by

Dr. Carl Hochsinger * of Vienna, who had pointed out that one of the diagnostic signs of the disease, when the metacarpal bones as well as the phalanges were affected, was that the swelling occurred chiefly at the distal ends of the metacarpal bones, *i. e.* in the neighbourhood of the epiphyses. This diagnostic feature was strongly marked in the present case.

The Chairman (**Dr. Frederick Taylor**) said he had followed the discussion with interest because he had had a case recently at Guy's Hospital in which a similar difficulty arose. It was a case in which a child, aged 10 years, had swelling of both knees, which was thought by some to be osteo-arthritis, although the occurrence of synovitis of the knees in congenital syphilis was recognised. She had a very firm, hard lump on the parietal bone of considerable standing; and it was in the history that her mother had suffered from syphilitic necrosis of one of the cranial bones. In the course of the girl's stay at the hospital she developed keratitis. She had no other indications of congenital syphilis and had not the appearance of a syphilitic child. The teeth were perfect. During the seven or eight weeks she was in the hospital there was no change in the condition.

Dr. A. A. H. Partridge (introduced), in reply, agreed that the question of rheumatoid arthritis had to be considered; each speaker had raised that question. He thought, however, that there were points of distinct differences between the two diseases, which he proceeded to mention. As to treatment, the child was an out-patient at an eye hospital, and till recently it had not been discovered that the joints were affected. One joint had recovered without treatment. As to the question raised by Dr. Parkes Weber, he thought the enlargement was due chiefly to the thickening of the tissues round the joint, and not to enlargement of the ends of the bones. There was some synovitis, no doubt, but that did not decide whether the condition was due to rheumatoid arthritis or syphilis. An attempt had been made to distinguish between the two conditions by the presence or absence of tenderness, but he believed that both were commonly painless. He did not see how any clear distinction could be drawn between the two except from the fact that the child was certainly syphilitic, but as both conditions were septic the joint changes in both might be much alike.

* 'Verhandl. d. Gesellsch. für Kinderheilkunde,' 1900, p. 47.

52. DIPHTHERIA OF THE ŒSOPHAGUS AND STOMACH.

By EDMUND CAUTLEY, M.D.

The specimens shown are illustrative of an unusual complication of diphtheria, and, in addition, there are several interesting points in the details of the case.

Edward F—, aged 18 months, was admitted into the Metropolitan Hospital on March the 28th, and died on April the 27th, 1903.

He was admitted under a surgical colleague for constipation and anuria of twenty-four hours' duration, preceded by an attack of diarrhœa and vomiting for two weeks. On examination the belly was found enormously distended. There was torticollis on the right side.

Under chloroform a soft rubber catheter was passed, but no urine was obtained. The rectum was ballooned. An enema was given, and from a pint to a pint and a half of liquid fæces were evacuated, and the child vomited much undigested food. The belly then became flaccid and he was much relieved.

Two days later I saw him, the question of the symptoms being due to the onset of meningitis having been raised. On careful examination a diagnosis of rickets and constipation was arrived at. The following is a brief abstract of the chief features of the case while under my care.

April the 7th.—Doing well. Constipation relieved by glycerine enemata. Has gained six ounces in the last four days.

8th.—Urine offensive, slightly alkaline.

10th.—Loss of three ounces in three days.

13th.—Gain of three ounces in three days.

15th.—The torticollis has subsided. Abdomen full and lax. Is cutting the upper canines; has twelve teeth. Urine offensive, alkaline, contains a slight cloud of albumin. Normal temperature.

16th.—Sudden rise of temperature to 100·8° F. in the morning and 103·8° F. in the evening.

17th.—Loss of seven ounces in four days. Stools undigested and loose. Urine muddy, offensive, specific gravity 1010, acid, contains trace of albumin.

21st.—Loss of five ounces in weight. Less fever. Stools very offensive, fairly digested. Urine contains albumin, casts, and numerous motile bacilli, but no pus or blood.

22nd.—Some nasal discharge. Tonsils enlarged and reddened.

23rd.—Languid and debilitated. Seems much more ill. Takes food badly, has vomited once; tongue a little furred; stools offensive and green. Widal's test, with a dilution of 1 in 30, caused marked clumping in ten minutes.

26th.—Vomited three times during the night. Bowels acted five times, the stools being green and containing curds. Urine acid, contains more albumin.

27th.—Steadily losing ground. More fever for two days. Occasional cough. Eyes turned upwards, to the left. Limbs somewhat rigid. Throat a little injected, and shows a small, dirty yellow patch, like exudation, on the left tonsil. Urine contains more albumin, granular casts, and many bacilli. Death resulted from failure of respiration in the afternoon.

The motile bacilli found in the urine proved on cultural examination to be a variety of the *Bacillus coli*.

Post-mortem examination (twenty hours after death).—All the organs pale. An excess of fluid in the serous cavities. Heart normal, undilated. A small ulcerated area was found on each tonsil. The larynx was normal, the lungs œdematous. The œsophagus was much engorged, thickened, very dusky in colour, and contained a considerable amount of adherent membrane in the upper third. The stomach was dilated, the cardiac end and fundus normal. Almost surrounding the smaller end, not quite reaching to the pylorus, was an intensely engorged hæmorrhagic area, due to acute inflammation of the mucous membrane, the size of a small female hand, partially covered with adherent

yellowish membrane. On the peritoneal surface were a few punctiform hæmorrhages. The spleen was dark, firm, and not enlarged. The kidneys were enlarged, the cortex thickened. The bladder was normal.

On microscopical examination of the stomach the cylindrical epithelium was found denuded, the tubular glands swollen, and many micro-organisms were seen on the surface, in and under the membrane; a few of them seemed club-shaped. The Klebs-Löffler bacillus was recovered by cultivation from both the œsophagus and the stomach.

Diphtheritic inflammation of the stomach is not common. It is, in fact, a rare disease, and is said to be always secondary to diphtheria of the throat or respiratory tract. It occurs almost exclusively in children, but cases have been recorded in young adults. It assumes various forms. Small isolated patches may be seen in the fundus, or streaks of membrane radiating from the cardiac orifice towards the pylorus. Extensive ulceration is unusual, and it is still more rare to find a universal covering.

Two good examples of the affection are recorded in the 'Transactions of the Pathological Society of London.' Edgar Willett * reported a case in a child aged one year. Tracheotomy had been done some time previous to death. The pharynx was affected; the œsophagus was normal. Nearly the whole of the mucous membrane of the stomach was covered with a fine, flaky false membrane. Soltau Fenwick † reported a case in a child aged three years. Tracheotomy was done twenty-four hours before death, and the child had been fed by nasal tube. The pharynx was not affected. Membrane extended from the larynx to the finest bronchial tubes. The œsophagus contained no membrane. The stomach was entirely lined with membrane, which extended for one-third of an inch through the pylorus.

Thus, in both these cases the stomach was extensively involved apart from any affection of the œsophagus. The explanation does not, therefore, consist in direct extension.

* Vol. xliii, p. 62, 1892.

† Vol. xlv, p. 60, 1894.

Probably the infected material is swallowed, or is pushed down by the tube in feeding, and the deficiency of hydrochloric acid in the gastric juice of the infant is a cause of the non-destruction of the infective material.

Many interesting questions arise out of the present case. The affection for which the child was admitted was somewhat anomalous. It was probably due to intestinal obstruction from unsuitable feeding after an attack of diarrhœa. Possibly drugs had been given to stop the diarrhœa, and had helped in producing the constipation. The suppression of urine was secondary to the intestinal condition. The cause and significance of the bacilluria are uncertain. A catheter was passed with the usual aseptic precautions on March the 29th. The urine was first noticed to be offensive ten days later. There were no indications of cystitis, such as pain or increased frequency of micturition, and the child was progressing favourably and gaining weight. The bacilli were found on microscopic examination at a later date, but may have been present from the start. Bacilluria is probably not uncommon in children, for it is frequently found on microscopical examination quite unexpectedly. A positive Widal's reaction, associated with bacilluria, suggested a possibility of enteric fever, but the history of the case, the course of the fever, and the absence of confirmatory symptoms enabled me definitely to negative such a diagnosis, even before cultural examination proved the bacilli to belong to the colon group. The absence of any post-mortem evidence of bladder disease, and the presence of casts, showed that the child had some nephritis. At the time the question arose whether there was a nephritis due to an infection by the *Bacillus coli*. The casts were found later in the illness, and were no doubt due to a diphtheritic infection of the kidney.

The association of the albuminuria with the diphtheria is somewhat vague. It was present though the temperature was normal. Possibly it was due to diphtheritic infection, occurring definitely sometimes before the rise of temperature of the terminal illness. The rapid loss of weight from

the 13th onwards is suggestive that it was on the 13th that the infection began to be active.

Both the mode of infection and the point of attack are uncertain. Not until six days after the onset of the fever was there any nasal discharge, and not until the same period was the throat found injected and swollen. At this time the left submaxillary glands were a little enlarged. This fact did not seem of much importance, seeing that they had been enlarged on admission in connection with the torticollis. Even on the day before death only a small excavated area was seen on the left tonsil. The nasal catarrh was very slight. The larynx, uvula, and soft palate were quite unaffected. Apparently the infection started in the œsophagus, and the stomach and tonsils were affected secondarily. Why the affection was localised in this way, and the exact manner in which it was acquired, must remain uncertain. The child was too ill to have toys, and seemed much more ill, from the time of admission, than the physical signs and symptoms warranted.

(*Exhibited May the 15th,* 1903.)

Discussion.

Dr. Theodore Fisher (Bristol) said he had now seen and made about 1700 post-mortem examinations, and had had a considerable number of diphtheria cases, but he had never met with a case like that of Dr. Cautley's. He had shown a stomach in a case of diphtheria before the Society about a year ago, which showed a condition which he thought was not so very uncommon. Small circular hæmorrhages were present all over the stomach, which he thought bore out the suggestion that toxins were secreted by the stomach in some diseases. If the urine were more frequently examined for bacilli they would probably be found in many cases. In the post-mortem room, if one looked for pyelitis one would find it in a large percentage of cases, and if the pus were examined under a microscope micro-organisms of the disease causing death might be found. For example, a few weeks ago they had a case of cerebro-spinal meningitis, and he remarked beforehand that in the kidneys they would probably find pyelitis, which they did. They also found some intra-cellular diplococci in the pus from the pelves of the kidneys, which were found in the pus at the base of the brain, and in the discharge from the base of the nose, and elsewhere.

Mr. Gordon Watson (introduced), referring to the bacteriological examination made, said that by the time he examined it the urine was practically acid, and there was no pus. It was crowded with motile bacilli, which he carefully examined by differential tests, to see if they were typhoid or not; and undoubtedly they belonged to the *coli* group. The section of the kidney showed commencing catarrhal nephritis. He also stained the kidney for bacilli, and found some, which he thought were *coli*. He could not make a diagnosis from seeing the section. With regard to the diphtheria, he obtained a pure culture of the Klebs-Löffler bacillus from the mucous membrane of the œsophagus and the stomach, and also in sections of the stomach wall, besides a large number of micrococci, many of which were streptococci. There were numerous club-shaped bacilli, which from their shape and grouping he thought might be safely put down to the diphtheria bacillus. The bacilli were found both in the fibrinous membrane and beneath it in the mucous membrane.

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53. A CASE OF ENLARGED BRONCHIAL GLANDS IN A YOUNG CHILD.

By J. PORTER PARKINSON, M.D.

This child, a male aged 4½ years, has been under my care for the past two and a half years. During most of this time he has suffered from a paroxysmal cough, attended with a marked "whoop" and frequently followed by vomiting. The cough has varied in frequency. During its worst periods, when, besides the other signs, there has been evidence of bronchitis in the chest, it has occurred nearly every half-hour, with two or three marked whoops during each paroxysm, and has, especially recently, been attended by vomiting, in the course of which a considerable amount of sputum was brought up. During his intervals of improvement he has only had about six or eight paroxysms of coughing, but during these the whoop has been well marked. The physical signs have varied comparatively little during the whole period of observation. He is a well-nourished

boy, but slightly small for his age. There is clubbing of the fingers and toes. There is some slight retraction of the right side of the chest, more marked below, and the costal angle is lessened on this side; the movements here are diminished, and there is retraction of the intercostal spaces during inspiration. The percussion note is slightly impaired, especially at the base. The breath-sounds are weaker on the right side, and in places somewhat bronchial in quality; there is no stridor, and at present no added sounds. The left lung is somewhat over-expanded. The heart is normal, its apex-beat being in the fifth space below the nipple. The abdominal organs and urine are normal. The sputum varies much in amount. During his last attack of bronchitis he brought up between half and three quarters of a pint each twenty-four hours. It is purulent, containing strepto- and staphylococci, and the *diplococcus lanceolatus*, but no tubercle bacilli. There are a few enlarged glands in the left side of the neck. The temperature during his attacks mounts to about 101° F., but at other times is normal. His weight is 2 st. 5½ lbs.

Though there is no direct evidence of their presence, I venture to suggest that in this case there are enlarged mediastinal glands pressing upon the right bronchus and interfering with the vagus nerve, hence causing some dilatation of the bronchi of the right lung, with accompanying fibrosis, and a cough attended by whooping, owing to the effect on the vagus nerve. This condition was present in a case I recorded in the 'Lancet' in 1898, in which the necropsy revealed the vagus nerve stretched by and adherent to the enlarged bronchial glands; while, owing to the pressure on the main bronchus, the bronchi of the left lung were dilated, and there was some collapse and fibrosis of the lung itself. The clinical aspect of the case was almost identical with the one now described.

(*Exhibited May the 15th*, 1903.)

Discussion.

The Chairman (**Dr. Frederick Taylor**) wished to hear some references made to the frequency with which such cases

occurred Years ago he had been very much impressed with descriptions of cases of enlarged bronchial glands, and with symptoms of tumour, and was led to believe they were very frequent. His subsequent experience had altered that opinion.

Dr. Porter Parkinson, in reply, would not express any opinion as to the frequency of such cases. He had been, perhaps, rather looking out for them since he made an autopsy of the first case, in which there was very definite enlargement of the bronchial glands. The vagus was absolutely flattened out by the glands. Since that time he had had an opportunity of making another autopsy, in which the condition was much the same. Whether the bronchiectasis or the enlarged glands was the primary condition he would not definitely say, but he thought it likely that the bronchiectasis was the primary condition. That was borne out by the fact that there was no tubercle in the gland; there was simply hyperplasia of the glandular tissue. He imagined it was more likely to be the secondary condition than the primary one. The question put to him was whether the bronchiectasis alone was sufficient to explain the very continuous whoop. It was so distinct that the child was not allowed by the mother's friends to associate with their children. She was sent out of hospital several times as a case of whooping-cough.

54. ATHETOSIS IN A CASE OF MITRAL DISEASE.

By FREDERICK TAYLOR, M.D.

Margaret H—, aged 15 years, by occupation a book-folder, developed scarlet fever when five years old, which was followed by diphtheria. She had rheumatic fever when six years old.

Present disease.—On March the 29th, 1903, seven days before her admission into Guy's Hospital, the patient woke up in the middle of the night, complained of great pain down the right side of the body, screamed, and was unable to move her right arm or leg. Some "twitchings" of the right arm and leg were noticed. Her speech was so much affected that her mother could not understand

what she said. She remained in bed the next day, with the same pain and in the same condition. In two days' time from the onset her speech was intelligible. She was able to walk, "dragging her right leg," and the movements of the arm were returning, except in the hand, which remained useless. Some involuntary movements of the hand and foot were noticed. The pain was less severe. She remained in this condition until her admission into the hospital.

On admission (April the 5th).—The patient is a pale but well-nourished girl. She complains of pain down the right side of the body, which cannot be definitely located. She gives her history clearly. Her speech is not affected, and her mental condition seems good.

There is a soft systolic bruit heard best in the mitral area, and traceable towards the axilla.

There is no complete paralysis, but there is weakness of the right arm, and still more of the hand. The grip of the hand is very feeble, and can only be maintained for a few seconds, when it relaxes. There is also weakness of the right leg. There is no paralysis or apparent weakness of the tongue or facial muscles. The patient states, however, that she has lost the power of whistling, which she formerly had. There are involuntary movements of the hand and foot on the right side, which occasionally involve the upper parts of the limbs. There are no involuntary movements of the facial muscles or of the muscles of the left side. On walking she slides the right foot along the ground. There are no alterations in sensation. The knee-jerks are brisk, and so are the superficial abdominal reflexes.

The pain in the side was not present for more than a day or two after her admission into the hospital, and was never severe.

May the 12th.—The patient's condition has not changed much, except that she has more power in the arm and legs, and the involuntary movements have become more marked.

The movements appear to be increased by observation, but not on attempting to use the hand. They are not present during sleep. When the hand is pronated and lying on the bed they are mostly confined to the wrist and metacarpo-phalangeal joints. There is occasional movement of the upper arm, especially when there is an involuntary supination of the hand. The movements at the wrist-joint are mostly those of flexion and extension, with lateral movement less frequently. There is not much tendency to rotation. There is no definite rhythm, but there are usually about forty movements of the wrist each minute. The thumb and fingers are hyper-extended and widely separated, the fingers being irregularly and independently flexed, sometimes only at the metacarpo-phalangeal joints, sometimes at the interphalangeal joints as well. The fingers are occasionally adducted. The hand is sometimes almost closed.

In the foot the sole is alternately inverted and everted, with movements of flexion and extension taking place at the ankle-joint simultaneously, so that there is rotation at the ankle-joint more frequently than at the wrist. There is no definite rhythm. The toes tend to be extended, but they are frequently flexed, and appear to move more uniformly together than the fingers.

The calf muscles can be felt to be contracting irregularly.

There are occasional jerky movements of the thigh, but the foot is not raised from the bed.

(*Exhibited May the 15th*, 1903.)

Discussion.

Dr. A. E. Sansom thanked Dr. Taylor for bringing the case forward. He would proceed very diffidently in dissecting and examining and criticising the case. He deferred to Dr. Taylor's superior judgment, but at the same time he thought there could not be any doubt that it was a rheumatic heart, and that there had been rheumatic endocarditis, whatever might be said about murmurs or the absence of murmurs. He might agree

with Dr. Taylor that it was a case of mitral endocarditis from so-called rheumatic causes. It seemed very clear that it was a case of embolism. There was plugging, and the plugging must have been of the left hemisphere, because it was a right-sided lesion. It must have been a large plug because in the first place there was not only paralysis, but aphasia. A good deal of it had recovered. How did a plug recover? Partly, sometimes, by softening and restoration of the circulation over an area previously disturbed. That was undoubted. Then, the centres for some of the movements were still impaired, and it might be there had been softening there. He would not use the word athetosis more than he could help, but there was probably an impairment of the motor tract. He was putting things clearly to himself, step by step. Then, they agreed first of all that it was rheumatic, and that there had been plugging. Plugging in a purely rheumatic case was not quite so common as people thought. It was very common in anything like septic endocarditis. In this case it would be agreed that there was multiple infection—there were sources of multiple infection. He was speaking of remote causes, not of present causes. There was scarlet fever followed by diphtheria, at any rate there was the possibility of multiple infection. It might be that infective endocarditis was not common in rheumatic endocarditis, but was common when there was a superadded infection. Some people thought so. It seemed to him, it was very possible that there had been something in addition to the ordinary endocarditis, something which had given rise to plugging, something which had caused embolic infarction, something which was extensive now, though less extensive than it was. They talked about the results of the nervous mechanism, and said there must have been an impairment of the sensory motor tract—he did not mind whether it were called post-hæmorrhagic chorea or hæmatosis, but at any rate it was an extensive impairment. Although it was smaller than it was, nevertheless the child would bear permanent symptoms, and probably there would be permanent hemiplegia. Dr. Taylor and he were not at war now about the first cause; they thought there was a rheumatic heart, and it was quite possible there was a superadded infection. As they were a society for the study of those things, it showed they were not there to express very positive opinions. He had tried to express opinions which were by no means positive under present circumstances. He wished to be pardoned for anything he had said over-positively. He thought it was a very extensive, a very severe lesion, which was likely to remain. He would not put any name to the nerve condition which had resulted.

Dr. Edmund Cautley asked if it were a common experience for athetosis to develop so quickly after hemiplegia.

Dr. G. A. Sutherland said the case was extremely interesting for them to see, and also from the point of view of diagnosis. Of course, the members were in the same position then as was Dr. Taylor when he first saw the case. Dr. Taylor was not quite sure then whether it was athetosis or chorea. Certainly, at first sight, the problem did present itself, and without being at all positive, his own impression was that it was a case of chorea, from the nature of the movements. Dr. Taylor had referred to slow movements. It seemed to him that those were very rapid movements for athetosis, and there was also the point raised by Dr. Cautley as to the very rapid onset of the movements. Then there was the association with endocarditis, which was, of course, a strong point in favour of chorea. The ordinary rheumatic endocarditis embolism was not a common thing in children, except in the very advanced cases. Then there was the hemiplegic distribution, which suited equally well for athetosis or chorea. The absence of the face movements was certainly against chorea, but he did not think it absolutely excluded the presence of chorea.

The Chairman (**Dr. Frederick Taylor**) said he was glad to hear Dr. Sutherland's doubt about the case, as it justified him in bringing it before the Society for inspection. At the same time he was not moved by Dr. Sutherland's criticism. Of course, he thought it was embolism; that was the only way in which he could attempt to explain it at all. As to the kind of movements, of course one could get choreic movements to a certain extent like them. He thought the movements of the foot were more violent than those of the hand, whereas it was common in chorea to have the movements of the hands more active; and he thought also this was more frequently the case in athethosis. The difference in rapidity in the movements did not appeal to him as a very strong objection. He said that the convulsive movements arising from lesions on one side of the brain might be very active, even of extreme rapidity. He did not know whether any members of the Society were familiar with a patient named Henry Lindey, who had had a "mad" arm for more than thirty years; his arm was continually in violent motion, so that it had to be strapped to his body. He dared not let his arm go for fear it should overpower him, so to speak. The man obviously had a brain lesion, for he had had an injury to his head some thirty-five years ago. There were cases, he believed, with all degrees of rapidity of movement between slow and fast. So far as chorea was concerned, sometimes it was fast and sometimes slow. Spasticity was not necessarily present in athetosis; it was replaced by abnormal mobility. The limbs were not perfectly paralysed; they had a certain amount of power; they were not spastic. When he spoke of chorea, he meant ordinary, Sydenham's chorea. He supposed

that the term post-hemiplegic chorea generally referred to a condition of athetosis. That might mean it was chorea to a high degree, and was post-hemiplegic, or at any rate post-embolic. He believed he would be right in saying that the first cases of athetosis described had no hemiplegia at all. Hammond's two cases of athetosis were cases in which there was a hemiplegic distribution of slow movement, without any definite hemiplegia. It was only afterwards it was recognised that movements of that kind, coming on after hemiplegia in children, were practically to be put in the same group. Even if it were somewhat rare to get cases quite like that, they had examples in the very first cases reported under that name. He would be glad to keep a watch on the case and report to the Society. Dr. Sansom had expressed the opinion that it was a permanent lesion, which was practically agreeing with him that it was athetosis. If it was choreic, Dr. Sutherland would naturally expect a recovery in the course of two or three months.

Postscript (July the 20th, 1903).—The condition of this patient is the same as when she was exhibited. The movements of the hand and fingers, and of the foot and toes, are constant, except during sleep. The facial muscles are entirely unaffected. There has been no improvement and no deterioration.

55. A CASE OF ACUTE ANTERIOR POLIOMYELITIS INVOLVING THE ABDOMINAL MUSCLES.

By FREDERICK TAYLOR, M.D.

Christine S—, aged 14 years, came home from school complaining of a headache one day in the first week of November, 1902. The next day she was worse; she was "light-headed," feverish, and had pains in the limbs. Three or four days later, on attempting to walk, her legs "gave way under her," and "her arms and legs became useless." The power returned in her arms after one month, but her legs remained "useless."

On admission (January the 24th, 1903).—The patient is a well-developed girl. There is extensive paralysis of

both lower extremities; the left leg cannot be moved except by the action of lumbar muscles shifting it in the bed; the right thigh can be rotated outwards and inwards slightly at the hip, and the toes can be flexed and extended. The muscles on both sides are wasted and flaccid, and the left gluteus muscle is in the same condition. There is no loss of sensation. The knee-jerks and superficial reflexes are absent. The muscles, when tested electrically, show the following results:

The rectus femoris, sartorius, quadriceps femoris, vasti, tibialis anticus, and soleus muscles on both sides show no reaction to the faradic or galvanic currents.

The left peroneus longus muscle shows reaction to neither electric current.

The right peroneus longus muscle does not act to faradism, but reacts to galvanism, KCC being > ACC.

In the arms the faradic current produces a contraction, except in the thenar eminences, and in the first interosseous space of the left hand. In the thenar eminences with galvanism ACC is > KCC.

The *abdominal muscles* are very weak. The recti cannot be felt to contract when the patient raises her head. Her cough also is feeble, and the only contraction visible occurs in the upper fibres of the right external oblique muscle, which then pulls the umbilicus upwards and to the right. The faradic current produces a good contraction of the right rectus muscle, especially at the upper part. With galvanism KCC is > ACC in all cases. (*Exhibited May the 15th*, 1903.)

Discussion.

Mr. A. H. Tubby asked if Dr. Taylor had had an opportunity of seeing the patient in an upright position, as it would be interesting to know what the curvature of the back was like. He said the great interest of such cases was where there was paralysis of the abdominal muscles alone. In the present case there was paralysis partly of the back and partly of the abdomen. The more he saw of these cases the more he found the trunk

muscles were involved, and he thought they were as much primarily involved as the limbs. It was difficult to find out the exact influence of the abdominal muscles in processes of equilibrium. He had been looking out for a case of paralysis of the abdominal muscles alone, but so far he had not found it.

Dr. Theodore Fisher (Bristol) said that Dr. Sutherland had shown one or two similar cases before the Society, where about two years after the attack one set of abdominal muscles seemed to be completely paralysed, so that when the child coughed or cried there was a great bulging, a regular balloon-out of one side. In that particular case the child died of broncho-pneumonia. He had the spinal cord in his possession, and there seemed to be a lesion of the upper part of the lumbar region. He was sorry that the sections had not yet been cut.

The Chairman (**Dr. Frederick Taylor**), in reply, thought that the lesion would have to be an extensive one to be readily observed. With regard to the bulging to which Dr. Fisher had referred, there was no obvious bulging in the present case. He assumed that in order to get bulging one must have a considerable portion of the muscles intact, and a limited portion affected. He had recorded a case of paralysis after herpes zoster. The muscles in the right and upper part of the abdomen corresponding to one or two intercostal nerves were paralysed, and when the other muscles were contracted, this portion was driven outwards so as to form a prominence. Where, as in the present case, probably three-quarters of the abdominal muscles were concerned, they would not be bulged by the remaining quarter which were in action. He had omitted to say there was exceeding weakness of the lumbar muscles. He was not surprised at Mr. Tubby's remarks. He was going to suggest the same thing. Probably they did not examine the abdominal muscles as much as the others.

56. A NOTE UPON A CASE OF INFANTILISM.

By E. C. WILLIAMS, M.D.

The photograph exhibited is that of a child aged 10 years. Her parents state that since four years of age she has not

grown to any appreciable degree. The father and mother are both well-grown and intelligent people. I have seen two sisters, who are of normal size. The child was first brought under my care at the Bristol Children's Hospital, because of her small size and lack of development. Her weight is 26 lbs. (average weight, 4 st. 6 lbs.) ; height, 36 inches (average

FIG. 26.—Patient, aged 10 years (on left), compared with child aged 8 years.

4 ft. 3 in.) ; circumference of head, $18\frac{3}{4}$ inches (average, $21\frac{1}{2}$ inches) ; length, 12 inches ; breadth, $10\frac{1}{2}$. The fontanelles are closed. The primary teeth are decaying. The child is naturally silent and slow, awkward to manage at times, and is rather dirty in her habits. She can read her letters and count. She answers simple questions fairly

intelligently. There is nothing to account for this condition; no heart lesion; no rickets; no congenital syphilis; no symptoms of pancreatic insufficiency. She digests cod-liver oil well. For a few weeks I gave her some thyroid extract; her mother thought she was brighter when taking it, but it had to be discontinued, as she lost 4½ lbs. in weight. Mr. Hastings Gilford,* in his paper on "Ateleiosis," a disease characterised by conspicuous delay of growth and development, divides his cases into three groups:

(1) Appearing before birth.
(2) During infancy.
(3) In the later stages of development.

The facial expression and general appearance of these cases must vary with the age of onset of the disease. The word infantilism has been used by many writers in cases where it is only a symptom, as in cases of congenital syphilis. Mr. Gilford, however, refers to a condition which he calls "normal infantilism," and says, "We recognise that growth may vary greatly, and that it may sometimes be excessive without being morbid; just in the same way as there are normal giants, so there may be normal dwarfs; and in some of these dwarfs there is not only delay or arrest of growth, but also of development. The chief point of difference between normal infantilism and ateleiosis is that development is arrested in a much lesser degree in infantilism than ateleiosis, and that these cases eventually may arrive at maturity. At this stage I do not think it is possible to say whether this child should be placed as an ateleiotic in group 3, or classed as a case of normal infantilism. If classed as an ateleiotic, she should certainly be placed in group 3, as the disease came on after the first dentition. Some cases of ateleiosis live to a good old age. With regard to the pathology of the condition, I do not intend to offer any suggestions. Whether the disease is a primary disease of the skeleton, or whether it is dependent on some abnormality of the sexual organs, pituitary

* 'Trans. Royal Medical and Chirurgical Society,' vol. lxxxv.

body, pancreas, or thyroid gland, is fully treated in Mr. Gilford's paper which I have already quoted.

(*Read May the 15th*, 1903.

Discussion.

Dr. George Carpenter suggested that Dr. Williams should make an examination of the uterus and Fallopian tubes and ovaries. There was no doubt that a certain number of males with congenital syphilis with atropied testicles never grew up; they were infantile for the whole of their lives. It would be interesting if a medical examination were made of the pelvic organs in the present case, and some report made as to their condition. It would make the case more complete if that were done.

Dr. E. C. Williams, in reply, said that a very careful examination had been made without avail to detect any disease of which infantilism might be a symptom. This was probably a true case of normal infantilism. There must be a distinction between cases where infantilism exists as a symptom, *e. g.* congenital syphilis and heart disease, and cases of normal infantilism.

57. NOTES OF A CASE OF SPLENIC LEUKÆMIA IN A CHILD.

By JOHN McCAW, M.D.

Isabella H—, aged 1 year and 8 months, was admitted into the Belfast Hospital for Sick Children under my care on February the 20th, 1903.

Family history.—She is the youngest of four children. The first child of the family was born at full term; it was apparently healthy and well developed, but died when three days old; no cause could be given for its early demise.

The second child—a girl—was born at full term, and was

well developed; she is now a strong, healthy child of five and a half years.

The third child was born at full term, and was strong and well developed. At the age of ten months he contracted whooping-cough, which became complicated with broncho-pneumonia and ended fatally.

The mother and father are living; both are strong and enjoy average good health; no history of syphilis could be obtained.

History of present illness.—The patient was born at full term and was healthy. She was breast-fed for one year and developed into a large and strong child. The present is her first illness. The mother states that the child was able to toddle about until three weeks ago, at which time she was observed to become weak in her limbs and to stagger much when walking. About this time she became drowsy and apathetic. A week later the abdomen was seen to be enlarged and the glands of the neck to be swollen, but she did not complain of pain. Dr. Hogg, the family physician, was consulted, and acting on his advice the child was brought to the hospital.

State on admission.—The patient is a well-developed and bulky child for her age; she weighs 1 st. 13½ lbs. No sign of either rickets or scurvy is present, and her appearance is that of a full-blooded rather than an anæmic child.

On examination the chest organs are healthy. The glands in the neck, in the axillæ, and in the groins are considerably enlarged; they feel hard, are freely movable one upon the other, and show no sign of softening; they are not tender on manipulation.

The abdomen is enlarged generally, which is due to the enormous size of the spleen, and also, but to a less extent, to the large size of the liver.

The spleen is easily visible as a large mass which moves up and down with each respiration. On palpation it is found to extend forwards to the umbilicus, while its free margin can be easily traced downwards as far as the pelvic

brim. It is very hard and dense to the feel, and is evidently very tender. Its surface is smooth and uniform, and the notch is very distinct.

The liver extends downwards for one inch below the level of the umbilicus, and almost meets the spleen in the middle line. It is not so hard, nor is it so tender on palpation as the spleen.

The kidneys are apparently normal in size. The urine presents a very peculiar appearance. It is cloudy when passed, but when allowed to stand until it becomes cold it is quite thick, and looks exactly like buttermilk. The first specimen I saw struck me as being chylous urine. The condition was found to be due to an enormous deposit of salts, among which numerous crystals of oxalic acid were found; there was also a trace of albumen. The reaction was intensely acid, the specific gravity was 1023, and the amount passed in twenty-four hours, 12 ounces. The temperature on admission was 98° F., and during her stay in hospital of one week no febrile movement was noticed, except on February the 22nd, when the temperature rose to 100·8° F.; it fell to 97° F. the following morning. Her blood was examined by Dr. Tom Houston, and the following is his report.

Red cells . . .	2,200,000 per c.mm.
White cells . . .	810,000 ,, ,,
Hæmoglobin . . .	39 ,, cent.

Differential Count.

Polymorphonuclear cells .	0·8 per cent.
Lymphocytes . . .	99·2 ,, ,,

The lymphocytes were true lymphocytes, and many of them showed signs of degeneration—irregular networks of chromatin of various forms. A few nucleated red cells were found in the films.

Two days after admission a few purpuric spots were observed on the legs and arms, and a day later a few bruise-like marks made their appearance, one over the left

eye, one on the upper left arm, and one on the right thigh. These did not increase in size, nor did any fresh ones appear. She never had any bleeding from either mucous or serous surfaces while in the hospital. During her stay she became very fretful, and cried incessantly, but no cause for this was apparent. She was taken home at the end of a week.

I was asked to see her with the family physician on March the 5th, when the following note was made:—She is now markedly anæmic and has lost in flesh considerably; her tissues are soft and flabby. Her temperature has remained about normal, and was 99° F. at the time of my visit. She is less fretful, but is tender over the body generally, and she cries when lifted or handled. No hæmorrhages have occurred with the exception that the motion passed on the previous day was streaked with bright blood. Several fresh bruise-like marks, about the size of a bean, are visible over the body, and on the right side of the chest and abdomen are numerous purpuric spots, and a few also are to be seen on the legs. The spleen is, if possible, larger, and now fills the entire left side of the abdomen, extending well beyond the umbilicus and down into the pelvis. It is very hard and resistent, and is evidently very tender, as any pressure upon it causes the child to cry out. The liver is about the same size as when last examined. The child died seven days later. From the time of my visit till her death she became rapidly weaker, and sank into a comatose state.

Dr. Hogg informs me that the breathing became very quick though no lung trouble was present, and that a considerable amount of cyanosis was developed. No hæmorrhages took place, nor was there any increase in the purpuric spots and bruise-like marks.

The spleen diminished in size very considerably before death, and was not nearly so hard to the feel. The lymphatic glands disappeared to a large extent, and they too were not so hard. It is to be regretted that no post-mortem examination could be obtained.

Treatment.—This consisted in giving arsenic in the form

of Fowler's solution. The dose was four minims three times daily, and this was rapidly increased until she was taking ten minims three times daily at the time of her death. It could not be said that any benefit resulted from its use.

Remarks.—The extreme fretfulness of the child during her stay in hospital was very remarkable. Was it due to pain in the bones the result of changes in the bone marrow? What was the significance of the diminution in size of the spleen and the lymphatic glands?

Read May the 15th, 1903.

58. SOME OF THE CASES SHOWN AT THE ROYAL ALEXANDRA HOSPITAL FOR SICK CHILDREN, BRIGHTON.*

1. *A case of Tumour of the Cerebellum.*

May G—, aged 6 years, admitted February the 28th, 1903, under Dr. Hobhouse.

Well up to four months previous to admission, when she began to have ataxic gait. There has been no vomiting and no pain in head. Gradually losing power in left arm and leg, and left side of face. Some paresis of left external rectus, and left pupil slightly dilated, reacts to light and accommodation, rather sluggish. Temperature usually above normal. Distinct inco-ordination of left upper and lower extremity. Some contraction of fingers of left hand. Speech and swallowing intact, but tongue points slightly to left. Left ankle-clonus. Knee-jerks marked. Double optic neuritis. Extension of toe well marked at times.

* Prior to the formal opening of the proceedings of the Provincial Meeting on June the 20th, 1903.

2. *A case of Tumour of the Peritoneum.*

Karl K—, aged 4½ years, admitted January the 27th, 1903, under Dr. Chaffey.

Abdomen noticed to be getting big three days before admission, when a well-defined sausage-shaped tumour could be felt extending across the abdominal cavity in the region of the transverse colon; not tender, and rather firm. General enlargement of abdomen, with some peritoneal effusion, which cleared up fourteen days after admission. There is an interval of tympanitic resonance separating tumour from liver dulness, which seems to be normal in extent. Lungs, heart, and urine normal. Spleen ? enlarged. Head rather square. The mother says he had rickets; she has had one miscarriage and one born dead; her father died of phthisis; no cancer in the family; says the boy fell and hurt his abdomen shortly before the swelling was noticed. The abdomen remains much the same in size, but the tumour is more defined and apparently larger. He walks about and does not complain of discomfort, and nutrition keeps good.

3. *A case of Mongolian Type of Idiocy.*

John A—, aged 3 years, admitted April the 20th, 1903, under Dr. Chaffey.

The mother says her confinement was natural; patient was born at the eighth month. The father and mother seem fairly healthy and intelligent; there is no idiocy in the family; they have always resided in Sussex. The child cannot talk, or walk alone; smiles when spoken to, and usually sits with the mouth open. The countenance is rather typical of the Mongolian type of idiocy, and there are well-marked curvings of the third and little fingers of hands. The thyroid gland can be felt, not enlarged. He has taken tabloids of thyroid extract for nearly twelve months without benefit, except some diminution in the amount of subcutaneous fat.

4. *A case of Hæmoglobinuria.*

Chas. M—, aged 2 years, admitted February the 19th, 1903, under Dr. Chaffey.

Has attacks about once a month, with pyrexia. Urine contains casts of hæmoglobin and urates after the onset of each attack. No cause assigned.

5. *A case of Enlarged Liver and Spleen with Anæmia.*

Arthur C—, aged 7 years, admitted April the 30th, 1903, under Dr. Chaffey.

Patient had measles two years ago. Cough for three months. Lungs normal. Temperature irregular, mostly subnormal. Pulse and respirations normal. There is considerable anæmia, and some pain on the right side of the abdomen. The liver can be felt below the costal margin, smooth, regular, and not tender. The spleen is evidently much enlarged, and rather hard.

Blood-count.—Erythrocytes, 4,110,000 per cmm.; leucocytes, 20,000 per cmm.; poikilocytes, a few; hæmoglobin, 42 per cent.

His anæmia is less, and his general condition has improved on tincture containing iron.

6. *A case of Anorexia Nervosa.*

Edward S—, aged 10 years, admitted April the 9th, 1903, under Dr. Chaffey.

His health is said to have been good till one of his sisters died, after which the patient seemed to be depressed in spirits, and began to refuse his food, saying he could not swallow. This occurred four months ago, during which period of time he has become greatly wasted. He lies and sits about usually, but can walk a little. Blinks his eyelids when spoken to, and answers questions reluctantly.

He drinks between two and three pints of milk *per diem*, but refuses nearly all solid food. There is no dysphagia now, and apparently no pain anywhere; the knee-jerks not obtained; no paralysis; no signs of organic disease of

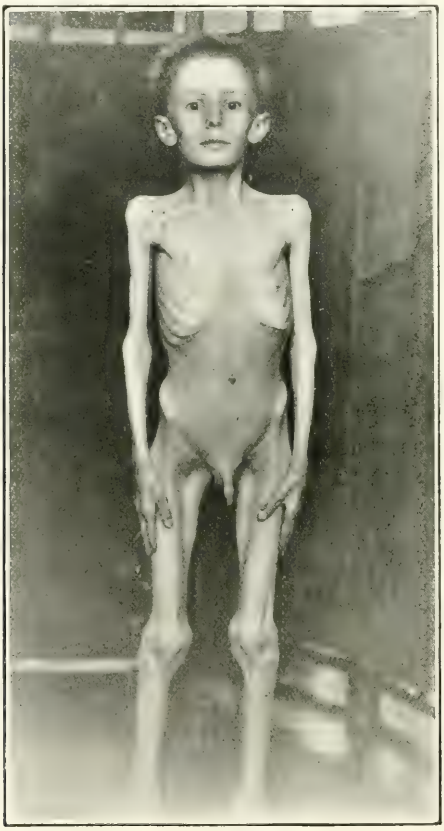


FIG. 27.—Edward S—, aged 10 years. Weight 2 st. 5 lbs. (Photograph by Mr. Basil Cook, house-surgeon.)

any organ. He has gained weight slightly since admission, but now scales 2 st. 5 lbs. only. He has improved somewhat on Tincture of Valerian, ten minims thrice daily.

7. *A case of Cerebral Abscess with Recovery.*

W. T—, aged 10 years, admitted May the 10th, 1902, under Dr. E. Hobhouse, for tuberculous meningitis. Otitis of the right ear for some months. Ill for one month before admission. Mr. Taylor opened a large abscess in the right temporo-sphenoidal lobe, a large hernia cerebri followed; but recovery is now complete after a long convalescence.

8. *A case of Actinomycosis, probably of the Pleura.*

G. G—, male, aged 12 years, under Mr. E. Hobhouse and Mr. H. H. Taylor. Admitted for empyema; abscess formed over the tenth and eleventh ribs on the left side posteriorly. When evacuated was found to contain typical granules, which microscopically showed the clubs and mycelium. The disease spread up the lung despite the administration of iodide of potassium up to 35 grains thrice daily, but has apparently become arrested since Röntgen rays were applied to the thorax.

9 *and* 10. *Two cases of Paroxysmal Hæmoglobinuria, one distinctly associated with Congenital Syphilis.*

A. B—, aged 6 years, male, and P. B—, aged 5 years, female. Under Dr. E. Hobhouse.

A. B— had well-marked nodulation of both tibiæ with considerable pain, which yielded to iodide of potassium. The history was simply one of repeated shivering attacks, but the mother said, on inquiry, that these were followed by passing dark water.

Cases of interest were also shown by Messrs. George Morgan, Bernard Roth, and Leonard Parry.

59. SOME REMARKS ON THE CLIMATE OF THE BRIGHTON DISTRICT.

By WAYLAND C. CHAFFEY, M.D.

My first duty is to thank the Council of the Society for asking me to preside, an honour I fully appreciate; and secondly, to congratulate the town and the medical profession of the district on the occasion of the third provincial meeting of this important Society being held in Brighton.

The combined towns of Brighton, Hove, and Preston may be said to be built chiefly on two spurs of the South Downs, having between them a valley, with Preston Park at the northern and the Old Steyne at the southern end. To the east of the latter the Downs rise somewhat abruptly, whilst to the west the ascent is more gradual. A good road passes along the front, a distance of several miles. To the west it is nearly on a level with the beach, and is protected for a considerable distance by high houses from the full force of the north winds. To the east there are really two roads: one on the top of the cliffs which is of considerable height; the other along the foot of the cliff, called the Madeira Road. In this latter situation there is also a well-constructed shelter.

At Hove, which is practically a continuation of Brighton to the west, the ground slopes very gradually upwards to the Dyke Road, which, at the back of the town, is 200 feet above the level of the sea. The Downs to the north and east are much higher, and extend across the country for miles in those directions. Ditchling Beacon is the highest point, viz. 858 feet.

With so many different aspects it will readily be imagined that the climate varies considerably according to the particular locality; and, speaking generally, it may be termed dry and bracing to the east, warmer and more equable to the west, whilst that of the valley between may

be said to constitute a mean, and to have more the character of an inland district.

The soil of the Downs on which Brighton and Hove are built is principally chalk, and, being of a porous nature, allows rapid absorption to take place after rain. This is a feature of great importance, whereby the air for miles round is rendered drier than is the case in most parts of the country. It is this fact, together with the absence of exuberant vegetation or marsh lands in the vicinity, that accounts for much of the salubriousness of the district. The Downs also serve as a natural barrier to the full force of the north and north-east winds which are prevalent during the spring of the year.

The influence of the sea on the climate of the coast is to render it equable. There is usually a cool breeze to be felt blowing from the south-west during hot summer days, and which is not experienced by those living many miles inland. During winter the proximity of the sea tends to render the climate of the coast warmer than that of inland districts.

The late Dr. Kebbell, in his excellent treatise on 'The Climate of Brighton,' 1859, defines the winter climate of Brighton as "mild, equable, dry and bracing," though in this latter quality it varies considerably in "different situations."

The mean annual rainfall and mean temperature of the air are much the same as in London, but there is a tendency for the ground to dry quickly after heavy showers.

The *water* is obtained from deep wells sunk in the chalk near the town. It is remarkably free from organic impurities, and is practically unlimited in amount.

The *drainage* is effected by means of a sewer which runs out to sea many miles from the town. By the adoption of modern sanitary methods, and of all means calculated to maintain the *mens sana in corpore sano*, the town authorities have greatly improved the place during recent years, so that, everything considered, it may now be said to compare favourably with any other seaside resort in the world.

With regard to the nature of the complaints mostly benefited by a stay in the town or its vicinity, I consider:

(1), pulmonary consumption, if not too rapid in its progress; (2), rickets; (3), some forms of anæmia; (4), atonic dyspepsia; and (5), general debility, are foremost; though no doubt many others are benefited more or less. I have shown to-day two cases of arrested phthisis, and could mention many others that have occurred during the last sixteen years. I have no doubt many other practitioners have had similar results, and I am glad to know that Dr. Newsholme, the Medical Officer of Health for Brighton, has, during the past year, treated many poor patients suffering from that disease at the Sanatorium on the Downs, and that he has met with encouraging success in this new venture (*vide* 'British Medical Journal,' May the 23rd, 1903).

Before bringing these few remarks to a close, I must remind you that Brighton, or Brighthelmston as it was formerly called, owes its popularity to Dr. Russel, who began practice here just 150 years ago. He attracted large numbers of patients to the town through his advocacy of sea water for various complaints, chiefly of a tuberculous nature. The book which he wrote and published in 1760 shows that he possessed a good knowledge of morbid anatomy, and is a very interesting work; but I cannot recommend his *sea-water cure*, especially the method of internal administration, considerable quantities having been drunk daily by his devotees. Probably we should do well, at the present day, to rely on the Mistura Alba, or Mistura Sennæ Composita (British Pharmacopœia), as subserving all useful purposes in the class of cases so graphically portrayed by the famous physician, whose name is justly identified with that of Brighton in the past.

(*Introductory address delivered at the Provincial Meeting, Brighton, June the 20th*, 1903.)

60. IS THE URBAN HOSPITAL TREATMENT OF EXTERNAL OR SURGICAL TUBERCULOSIS JUSTIFIABLE?

By A. H. TUBBY, M.S.

We are all agreed that the object of the treatment of disease is to cure, as quickly and as painlessly as possible; and if this be effected, it should be of such a nature as to enable the patient to earn his or her living subsequently. Further, the cure should in the case of an infective disease —and tubercle may be fairly considered such—render the child a healthy member of the community, and *ipso facto* no longer a source of danger and infection to others.

The question at issue is, whether the treatment of tuberculous disease of the bones, joints, glands, and mucous membranes, which we for convenience designate by the term surgical or external tubercle, ought or ought not to be undertaken in the hospitals of cities and towns; and if so, does treatment carried out in these places fulfil the criteria which have been mentioned?

In order to examine this question I have been at the pains to collect statistics for the year 1901 from four children's hospitals in London, and from one in the country. In response to my appeal to the London hospitals, four alone replied, namely, the Belgrave Hospital, the Evelina, the Paddington Green, and the Victoria Hospital.

I was enabled to secure returns without difficulty from one country hospital, the Sevenoaks Hospital for Hip Diseases, so that it has been possible to obtain a comparison between the results of treatment in town and country; but it must be clearly stated that the conditions which obtain in the two groups of hospitals are not identical. A children's hospital in London is maintained for the treatment of all kinds of disease, and the natural wish of the administration is to obtain as many satisfactory results as may be in a given period. Therefore acute cases of every kind of disease,

capable of rapid treatment, are often preferred to more chronic ones, such as the tuberculous; and even if the latter are admitted, it is more often with a view to immediate operation than not, and when operated upon such cases are frequently sent out before they are fully healed, so as to make room for more acute, and to many people more interesting cases. In fact, the stress and strain upon children's and general hospitals in London is such, that it is not possible to give that adequate and prolonged attention to tuberculous disease of the bones and joints which is an essential factor in the success of the cases. Presuming that they could be retained for a considerable time, there is no doubt that the air of a London hospital is not the best for them. Often I have noted a temporary improvement in a child's condition after admission, due to rest, good food and cleanliness, and for a week or two there has been seen some betterment consequently on the removal of a breaking-down focus of disease; but if such cases are retained for any length of time the signs of improvement disappear, and they steadily retrograde. Then, as a desperate resource, they are sent off for a short time to the country or sea-side, where they may or may not heal, and where they are a focus of infection to other patients just recovering from acute illnesses. Eventually they drift back to London, and begin the weary hospital round over again.

Now, in the hospital entirely devoted to the treatment of surgical tuberculosis there are no conflicting interests arising, no other and more acute cases to be considered, and therefore every one works with one object in view, namely, the cure of cases—however chronic they may seem. They are taken in with this object alone, and are retained until they are well, or it is evident that they are entirely beyond relief by any of the present means in our power. Herein lies the essential difference between hospitals where mixed cases are taken in and those devoted to one object alone. The general want of the latter is a radical defect in our arrangements.

In compiling the statistics, inquiries were directed to the following points, and the result has been carefully tabulated. The age and sex of the child have been noted, and in this paper children exclusively are dealt with, namely, boys under the age of ten years, and girls under the age of twelve years. Then the duration of treatment previous to the last admission into the hospital from which the returns are made has been ascertained; also the number of admissions to other hospitals, because we find that many children go from hospital to hospital, while some few return again to the same hospital. The duration of the stay in the hospital on the last occasion is also given, together with the total number of operations performed from first to last, and the results under three headings.

1. *Relief;* meaning by this that the patient's condition has been somewhat ameliorated by a stay in a hospital, but that the disease has not been entirely cured, as is evidenced by the discharging sinuses, infection of the skin, or the incidence of other tuberculous lesions.

2. *Cure;* that the disease has subsided and healed, and that there are no signs of active tubercle anywhere; and, further, that there is a reasonable probability of the child being able to gain a livelihood, or at any rate that he has ceased to be a source of further infection to himself or a focus for other people.

3. *The number of deaths* have been noted in the hospital. The numbers of these given must be necessarily smaller than actually occur, because many of the so-called "relieved" cases are lost sight of, and a fatal result follows eventually from phthisis or tuberculous meningitis. Too often the child lingers on and dies at home, having spread infection far and wide, or drifts into a hospital, the statistics of which have not been available to us.

To refer to actual details, we find that in the urban hospitals mentioned 218 cases were admitted, being 115 females and 103 males, and they were of the average age of five years and eight months, a very tender age to be the victims of chronic trouble such as this, and they had under-

gone treatment for sixteen months previously. Forty-five were afflicted with tuberculous glands in the neck, 10 had tuberculosis of the skin, 90 of the joints, and 64 of the bones. The minimum stay in the London hospital was five days and the maximum 176, the average being forty-one days, or nearly six weeks. On these 218 patients 415 operations were done; so that, taking the average, about two operations were performed on each patient, and in one case it is recorded that he was operated on as many as eighteen times, another twelve, and another eleven; and these operation statistics constitute to my mind a very strong indictment of the urban hospital treatment of tubercle. It is a moot point to me whether operations on the bones and joints of children in city hospitals for tubercle are not productive of as much harm as good, in the way of further spreading the disease by infecting fresh tissues. However, as the result of all this expenditure of labour and time only 68 of the 218 cases, or 31·6 per cent., were cured; 128, or 58·6 per cent., were relieved—and relief is no cure in tubercle, for half-cures are worse than none; in 13 cases the result was doubtful or unchanged; and 9 patients are known to have died in hospital.

Now we turn to the statistics of a country hospital—the Hip Hospital at Sevenoaks; and here it must be premised that, under the present chaotic arrangements for the treatment of tubercle, this hospital does not have a fair chance with all the cases from the commencement, and this is, as I shall insist upon later, an essential point. A considerable number of their patients are sent down from London hospitals after operative treatment has been tried and failed. In one case three operations had been done, and the remark in the note-book is to the effect that "the result is not one to be proud of." I need not weary you with the repetition of all the statistics, but give merely the salient points. Forty-two cases were under treatment in one year; the average age was a little over six years. Thirty were cases of joint disease, 10 of the bones and spine, and 2 suffered from enlarged glands. The average

duration of their stay in hospital was ten and one-third months. The total number of operations was 3, and the results show 14 cures; 24 were relieved, and many of them are progressing most satisfactorily and promise thoroughly well. Three were discharged incurable, and one died of tuberculous disease of the kidney.

Now, it will be noted that in the case of this hospital the duration of stay was forty-five weeks, as compared with six weeks; the proportion of cures was greater than the London hospitals can show, while the operations performed were represented by the small number of three—that is to say, the percentage of operations in the town hospitals, as compared with that in the country hospital, is as 19 to 0·07, or nearly thirty times as many. There can be no doubt as to the conclusions to be drawn from these results.

As to cure.—The number is by no means so great in town as in the country, nor is the cure so complete, for the number of relapses is far greater, as is shown by patients continually going the round of the hospitals. The urban hospital treatment cannot therefore be economical, meaning by this term that the best results are being obtained for a given expenditure of time, money, and energy; and under the present disjointed arrangements, whereby country hospitals are compelled from sheer pity to take the cases that have failed in London hospitals, the resources of both are wasted. It is true that cases remain longer in hospital in the country, but the results are infinitely better.

Then we come to the question of the *painlessness of the treatment.* There can be no doubt on this point when we contrast the number of operations, and I am disposed to say that the enormous number of operations done in London, as compared with those in the country, is a very grave state of affairs, and needs urgent consideration. For, after all, it must be remembered that many of the operations on bones and joints for tubercle are very far from being such successes as they promise when they are undertaken. There are no operations which need more careful consideration as to the after possibilities than those for tuberculous disease of

the bones and joints. Everything looks so well on the operating table, and it appears as if the disease had been removed, but the disappointments are many and great. More particularly is this so after excision of the hip and knee. We can all call to mind a large number of limbs shortened or twisted, and therefore useless, and with many recurrences of tubercle in the soft parts, followed by sinuses and scrapings. Above all, we meet from time to time cases of hip-joint trouble in which it is necessary to amputate, and it must surely seem that the removal of a fourth part of a patient's anatomy for a curable disease is, under any circumstances, a grievous reproach to modern surgery. After considerable experience, I cannot help expressing the opinion that better results are obtained in those cases which are treated by rest from the first, and where there is no excision or ablation of bone and wide cutting of the soft parts.

We will now pass on to the suggested remedies.

1. *The place of treatment.*—Tuberculous children should not be put into hospitals in cities or large towns, but properly equipped establishments should be started in the country or at the sea-side. The increasing means of communication will easily meet the difficulty of the attendance of competent surgical officers.

2. These hospitals should be devoted to one object only, just in the same way as sanatoria exist for the open-air treatment of phthisis alone. If internal tubercle is treated in special hospitals, why not external?

3. The rural hospitals must be conducted on definite principles. There should be no limit as to age, the youngest children being equally eligible with the older. In fact, the younger the child, the more hopeful is its case, and the more urgent is it to take it in hand at once and thoroughly. Cases once admitted should stay until they are cured, or clearly proved to be incurable, and for the latter separate establishments should be provided, so that they cease to be a danger to the community. Cases which are undergoing cure should not be liable to interruption, owing to the want

of subscribers' letters or other red-tapism, and a case once entered upon must be followed right through.

Now, to obtain this ideal state of affairs, the present chaotic and wasteful absence of system must be abandoned. The first essential is to have a central council to deal with the whole matter and with cases as they arise. Every case applying for relief to a city hospital should be notified to the central council, who also administer the rural hospitals, and the cases should be placed in some one or other of them, careful notes of the progress being made. And after the child has left the hospital examination should be carried out from time to time, and if recrudescence occurs, the patient should be sent back to the particular hospital at which he was treated, and as far as possible placed under the same surgeon, with the object of securing a continuity of treatment under one skilled observer—a most important factor in success, and the absence of which is so striking at the present day. Until a central board is established, with powers such as have been mentioned, it is to be feared that external tubercle will go on increasing.

An important question is *as to funds*. While some hospitals have their own convalescent homes, where they mix indiscriminately tuberculous and non-tuberculous cases, many others have none. It is found on inquiry that one child in every ten admitted to a children's hospital is the subject of external tubercle. Surely it would be better and more economical for such hospitals to close one tenth of their beds and earmark one tenth of their income for the purpose of establishing branch hospitals. At any rate, they would turn over the remaining nine-tenths of their beds quicker with the more acute cases of disease. If it should happen that the amount which each hospital can contribute is too small to establish a separate branch hospital, then there is no reason why several committees should not combine, and have beds proportionate to their contributions.

But the gist of the whole matter appears to me to be the need of a central organising committee, all the members having an intelligent appreciation of the drawbacks of the

present arrangements, and an earnest desire to deal with the question in a thorough and business-like manner.

Read at the Provincial Meeting, Brighton, June the 20th, 1903.)

Discussion.

Mr. R. Clement Lucas remarked that there was so much to be said on that great subject that it was impossible to make any comments which would be of service, either in assisting Mr. Tubby to carry out his scheme, or in regard to the hospitals which the author so much condemned, and which carried out the treatment of tubercle in London. He would only make the suggestion that, in his experience, it was not so much because the cases had been operated upon for tubercle of the joints in London which accounted for the second break-down, but that the patients returned to their unhealthy surroundings. There was not time to enter at all adequately into the question, but all would agree that country and sea air, at such places as Brighton, was of the greatest assistance in eradicating tubercle when the disease had been once put on a fair road to recovery.

Dr. A. E. Sansom wished to testify his admiration of the paper read by Mr. Tubby, which was worthy of the author and of the Society. It was a genuine attempt to grapple with a very difficult subject, and he regarded that subject as the care and management of tuberculosis, because he could not dissociate the surgical aspect entirely from the medical, and the ideas enunciated applied to both branches of the profession. It seemed rather a confession of feebleness when one attempted to treat in London tuberculous disease without bringing to bear fresh country air, and he used the word "country" advisedly. Country air was a most important therapeutic factor. Of course, oxygen was available in London, and one was ready to believe that lives had been saved by pure oxygen inhalations—*i. e.* fresh air rendered purer by means of oxygen,—but it was of nothing like the value of fresh country air in the treatment, as shown by Mr. Tubby's paper. He thought a very valuable and important scheme had been enunciated, and he thought the general directions given in Mr. Tubby's paper would bear great fruit. So far as one was able to judge at the present time, tuberculosis was a slowly manifested disease. Although they might agree it was an acute disease, he thought there was something against the acceptance universally of the bacillus theory alone as to the causation of the disease; and he was not sure that one was not a little too urgent in one's recommendations whose object was to prevent other people breathing into the same air and getting a bacillus into their bodies. But, however

that might be, nothing would ever convince him against the view, based upon a long experience, that fresh air—country air—was of immense therapeutic value in the condition. Therefore he was quite willing to receive the evidence which Mr. Tubby had brought forward, to accept it to the full, and to state that it was one of the most important public questions that could come before them. He pleaded that the medical as well as the surgical aspect of tuberculosis should be considered, and that Mr. Tubby's evidence was quite clear as to the therapeutic efficacy of fresh country air. The subject had been thoroughly well treated, and was thoroughly at one with the purposes of the Society.

Mr. A. H. Tubby, in reply, said there were few questions which he need answer, because it was not a subject admitting of much discussion, as it was really proven. The object he had in reading the paper was to bring the conclusions home by facts. Every one who had worked at a children's hospital would admit that the case was proved *primâ facie;* but one wanted to know how far one was wrong in operating upon individual children in London hospitals. He confessed his experience of eleven years at the Evelina Hospital, and other places, had convinced him that it was not desirable to operate on the cases under discussion in towns. In fact, he was not disposed to operate, except in cases of grave urgency, on the condition in any town hospital, for the reasons he had stated.

Mr. Clinton Dent said the issue raised by the paper was of such importance that it would be a very great pity if it were lost sight of in the mass of work which the Society had to get through that evening. He suggested it would be well to appoint a sub-committee of the Society to consider the matter brought forward by Mr. Tubby, and report to the Society. He believed a great deal of good would come from such a course. The sub-committee might say, first, whether the scheme was a sound one; and secondly, whether it was a practical one. He therefore proposed that a sub-committee be appointed to consider the question.

Mr. Francis Jaffrey seconded the motion, and it was unanimously adopted,

The Chairman (**Dr. Wayland C. Chaffey**) (Brighton) asked Mr. Tubby to nominate the gentlemen whom he would like to serve on the sub-committee.

Mr. A. H. Tubby, in response, thanked the meeting for the interest shown in his paper. He believed it was the kind of work which should be undertaken by the Society, in addition to the ordinary routine work of exhibiting cases and reading papers. One rather looked to such a Society to do administrative work. He suggested that the sub-committee should consist of Mr. Clinton Dent, Dr. Dawson Williams (the Editor

of the 'British Medical Journal'), who had taken the greatest possible interest in such questions, Mr. Sydney Stephenson, and Mr. Francis Jaffrey; and he would also be happy to serve on it himself, if permitted.

The names were put to the meeting, and duly approved.

61. TIN MOUTH-ORGAN OR WHISTLE REMOVED FROM THE JEJUNUM BY LAPARATOMY THIRTEEN DAYS AFTER HAVING BEEN SWALLOWED.

By R. CLEMENT LUCAS, B.S.

T. C—, aged 3 years, was admitted into Guy's Hospital under Mr. Lucas's care on April the 7th, 1903. He had been playing with a tin mouth-organ or whistle, which is placed between the teeth and lips when in use, and had swallowed it. He was brought to the hospital shortly afterwards and skiagraphed, when it was discovered just to the left of the median line, and thought to be at the pyloric end of the stomach. As there were no symptoms of importance it was determined to wait and give it time to pass through the intestine.

He was skiagraphed on April the 9th, the 15th, the 18th, and the 20th, and little or no alteration was discovered in the position of the foreign body. It appeared to be opposite the lower part of the first lumbar vertebra on the left of the median line, and above and internal to the tip of the ninth left rib.

Whilst under observation the child had no sickness or elevation of temperature, took his food well, and passed normal motions. On April the 20th, thirteen days after the accident, Mr. Lucas determined to perform laparotomy and explore the abdomen for the foreign body, which was still believed to be lodged in the stomach.

A.C.E. was administered, and an incision made about 3½ inches in length over the left linea semilunaris. The outer edge of the rectus muscle seen through its sheath was exposed by the first incision. The aponeuroses were divided in this line external to the sheath. Two or three small vessels were twisted and the peritoneum opened. The stomach was readily brought into view and pulled up into the wound. It was carefully explored from the outside by being passed through the fingers from the pylorus, which was distinctly felt, to the œsophageal extremity, but no foreign body could be detected within it. Having satisfied himself that the foreign body was not in the stomach, Mr. Lucas put his hand in the abdomen and felt for it among the intestines. Lifting up the transverse colon and omentum, he discovered the foreign body in the small intestine, a loop of which containing the whistle he drew up through the wound. It lay stretching the intestine, one edge towards the mesenteric attachment, the other towards the free edge, rendering the intestine so transparent that the body could readily be seen through the intestinal walls. A longitudinal incision about ¾ths of an inch in length was made over the edge of the instrument, when it quickly slipped out by pressure from behind. A little yellow intestinal fluid followed, but the abdominal aperture and the loop of intestine having been packed all round with sterilised gauze, none of the contents of the intestine was extravasated into the peritoneal cavity. A continuous fine silk suture perforating all the coats was used to close the aperture, and a second continuous Lembert suture to invert the first. The intestine having been replaced, a continuous fine silk suture was used to close the peritoneum, interrupted buried silk to bring together the muscles, and a continuous horsehair suture for the skin. Cyanide dressings were used.

The child was fed by nutrient enemata for the first three days, a little water only being sipped by the mouth.

On April the 23rd the wound was dressed and found to be healing well. The patient was happy and contented. His temperature was 98° F. Milk was allowed.

On April the 24th custard and eggs were ordered.

On April the 25th he was given bread and butter pudding and beef tea.

May the 1st full diet was ordered.

May the 5th he was discharged, cured, on the fifteenth day after the operation.

The temperature never rose above 99° F. throughout the convalescence, but was as low as 97° F. on the day after the operation. The mouth-organ or whistle removed is circular, measures $1\frac{1}{8}$th of an inch in diameter, and is composed of two convex pieces of tin united at the edge. There is a circular hole in the centre measuring two lines; and in thickness the organ measures $\frac{1}{2}$ an inch. The two pieces of which the organ is composed had been displaced one over the other so as to leave a rough sharp edge at the margin, and an aperture on one side.

Remarks.—The two chief points illustrated by this case are, *first*, the certainty in diagnosis as to the presence of the foreign body determined by the use of the X-rays; and *second*, the ease and safety with which such bodies lodged in any part of the alimentary tract may be removed.

It is true we were deceived as to the position of the tract in which we were led to believe the foreign body was situated, but we knew for certain that it was lodged high up in the abdomen. Having explored the stomach from the outside without success, we were not long in discovering the foreign body in the upper part of the intestine, and then its removal presented no difficulty. In the case which I brought before this Society two years ago of a nail lodged in the second portion of the duodenum, the X-rays determined with exactitude the portion of the intestine in which the foreign body was lodged, as the vertical fixed position of the nail on the left of the spine could scarcely be maintained in any other part of the intestine. In that case the diagnosis was more exact, but owing to the great depth of the second portion of the duodenum the operation presented greater difficulties. In the present case the foreign body once discovered gave us no anxiety, either at

the time of its removal or afterwards. The patient showed no discomfort, and only cried for food when the other patients were being fed. His temperature, at first subnormal, never rose above 99° F., and his convalescence was uninterrupted.

In making a diagnosis of a foreign body in the stomach by means of the X-rays it is fair to say that in this case one condition was absent, viz., that the body should alter its position when the patient is turned from side to side. But the photograph showed it fixed so high up and so near the median line that we came to the conclusion that the organ had become entangled edgeways in the pylorus, or at least that it could not have passed further than the duodenum. An absolute fixation for a period of nearly a fortnight at one spot in the jejunum is probably to be explained by a portion of one of the valvulæ conniventes having become caught in the split edge of the whistle.

A skiagram of the foreign body in position in the abdomen, and the mouth-organ removed, were exhibited.

(*Read at the Provincial Meeting, Brighton, June the 20th,* 1903.)

Discussion.

Mr. Clinton Dent said he had a case, also in a child, with precisely the same sort of foreign body in its inside. For some time it was impacted in the œsophagus, where it was seen by means of X-rays. An anæsthetic was given, and he attempted to remove the body from the lower part of the œsophagus with an ordinary hook. The gentleman in charge of the X-ray department saw the hook go down and catch on to the whistle, and saw it rise up until it reached the mouth, when it was missed. Subsequently it was apparently in the stomach, but a few days later it had shifted its position in the abdominal cavity. After about ten days it became fixed in one position, and remained there a considerable time. Anxiety was felt lest it should cause ulceration. He could not see it well by means of the screen, but the X-ray gentleman was positive about it. He (Mr. Dent) performed laparotomy, and searched the alimentary tract from the cardiac end of the stomach to the rectum, but he could not find it. The result of the operation was the disappearance of the whistle, though he could not say how. It had never been observed in the stools, and it was now some months since the occurrence. He felt no doubt that it really passed

along the intestinal tract and was evacuated with the motions. He was congratulated by the parents on the extraordinary skill with which he performed the operation, and the success which attended it.

62. THE FUNCTIONS OF CHILDREN'S HOSPITALS.

By EDMUND HOBHOUSE, M.D.

I HAD proposed to say something to-day about some of the problems which diseases of children present to us, but I found that there was already a great deal of material for clinical discussion on the agenda, and I propose therefore with your permission to say a few words about the functions, or perhaps I should say possible functions, of children's hospitals in general.

I do not suppose that any one can see much of the work of a hospital for children, at all events in a large town, without wondering how it is that any reasonable proportion of the population grow up to a decent measure of health and strength. I may be a pessimist on this point, but I must confess that I am often appalled at the number of children suffering from wholly preventable disease and the effect of this on the future of the population of this country, most of the mischief being done during the first two or three years of life.

The fundamental causation of by far the greater proportion of children's diseases may be summed up in three words—ignorance, poverty and neglect, the outcome of which are bad air, bad food and bad clothing. It is difficult to say which of these three is the most potent factor in disease production, but rickets is typically the disease of bad diet, tuberculosis of bad air, rheumatism and chronic diarrhœa of bad clothing; all co-operate. Perhaps bad food is the worst, since a well-nourished child may be able to resist the effects of bad air and clothing, whereas an ill-fed rickety infant falls an easy prey to every disease.

When we consider the whole question of preventable disease, the matter is bad enough in general hospitals, but it is ten times worse in those for children; and it must be remembered that whereas adults are to a large extent the victims of their own folly, the children are wholly irresponsible for their own misfortunes.

If we could but eliminate from child life merely those diseases which we know to be readily preventable, we should, besides reducing our own hospital work about fifty per cent., also remove a vast amount of that disease in adults which is merely the sequela of disease in childhood; such as the later forms of tuberculosis, cardiac disease, a certain amount of epilepsy, and much skeletal deformity, and a vast amount of intangible physical and mental debility which result from rickets.

We should all admit, I suppose, that rickets and tuberculosis furnish a large proportion of our cases; I should be sorry to say what proportion, and yet both, especially the former, are eminently preventable diseases; so to a certain extent is rheumatism, which furnishes another large class of cases and produces in the form of valvular disease perhaps even less remediable results as regards the future of the child.

As regards rheumatism, we are of course at present in ignorance of the way in which infection (I use the word deliberately) takes place, and therefore can do less to prevent it, though proper clothing has no doubt an important influence. It may be hoped that in the light of our present knowledge it will not be long before the channels of rheumatic infection are known and prophylaxis will become easier.

All these things are perfectly familiar to all of you; I am merely repeating an oft-told tale when I recall them; but I should like to ask whether we act on them as fully as we might. If there is one thing in which preventive medicine is of more importance than another, it is in children's diseases; the loss of life is bad enough, but the permanent crippling of an enormous number of lives and the stunting of so many

more is to my mind far worse. Happy the child who dies early, rather than survives to be the victim of chronic hip disease or some valvular lesion of the heart.

So far as is possible, I should like to see all children's hospitals and lying-in hospitals become centres of education for mothers. It is true, of course, that not very much can be done, and that only a small proportion of the population will be affected; but something may be achieved by the constant distribution of leaflets, such as we have here, reprinted by kind permission of the Medical Committee of the Great Ormond Street Hospital, who originally issued them some years ago.

In this way a certain amount of knowledge of the elementary facts as to the rearing of children is disseminated. Is it not the duty of this Society, which is the only society for the study of children's diseases in Great Britain and Ireland, to take up seriously the question of the prevention of children's diseases and to consider how far it is possible for children's hospitals, perhaps in co-operation with the municipal authorities, to become centres of enlightenment for the poor population?

When one considers the present condition of a large proportion of our urban population and the fact that the country districts are still being steadily depopulated, whilst the inhabitants are flocking into the already overcrowded towns, the prospect becomes appalling. It is on the children that the stress of town life really falls most heavily, it is they who chiefly bear the burden of the struggle for existence, and we are brought face to face with this fact every time that we visit our hospitals. The results of the mother's ignorance, of the father's drunkenness, of the deficient milk supply, of the commencement of education on babies who are scarcely out of their cradles and its termination with children not halfway through their teens, are constantly before us.

I venture to think that there is no single social or economical problem which can compare for importance with that of the proper bringing up of the young children, seeing

that on it depends the future of our race; and that we, who have the fullest experience of the results of the present state of things, ought to lose no opportunity of impressing on the proper authorities the enormous evils which are now rife.

We might as a society discuss these matters in their wider bearings, and get suggestions from members in all parts of England as to the best remedial measures.

To take the question of food first, which is perhaps the most important of all and in some ways the most easily remedied. I think the medical authorities of children's hospitals might well approach the municipalities and point out to them the importance of a *proper milk supply*—in these days of separated milk a much more difficult question than it used to be.

If a proper milk supply for infants up to two years of age were ensured, I believe that the whole aspect of much of our population would be changed in a generation. It could be done if a mere fractional amount of the enormous drink bill of this country was spent in giving milk to the children instead of beer and spirits to the parents.

I think I am right in saying that the Municipality of Glasgow (only following in this respect the example of the New York authorities) have taken steps to ensure a pure milk supply to all infants; and the question ought to be considered by all municipalities.

The question of bad air is largely in the hands of those authorities who are entrusted with the supervision of insanitary and overcrowded houses.

The question of clothing rests chiefly with the parents themselves, with the result that certainly not one child in ten of those who appear in the out-patient room is really properly clothed, as regards quantity or disposition round the body.

I am very far from saying that we can produce a revolution in these matters whatever efforts we make; all I wish to urge is that we should not consider them as outside the scope of our inquiries, whether as a society or as

individuals, but that by the dissemination of suitable literature on all these questions, by co-operating with the various local authorities, and impressing on them the urgent need of reforms, and lastly by preaching ourselves when opportunity offers, we should do what we can as Englishmen as well as doctors to assist in the solution of these tremendous problems.

(*Read at the Provincial Meeting, Brighton, June the 20th*, 1903.)

63. AN OPERATION FOR THE REMOVAL OF TUBERCULOUS GLANDS FROM THE ANTERIOR AND POSTERIOR TRIANGLES OF THE NECK THROUGH AN INCISION IN THE HAIRY SCALP.

By R. H. PARRY.

The object of this paper is to show that masses of glands widely distributed in the neck may be removed by operation without leaving a visible cicatrix.

It may be as well to indicate at the outset the types of cases in which the operation has been carried out.

The first of the types is that in which the glands below the angle of the jaw are enlarged, then those adjoining and lying beneath the sterno-mastoid muscle, and finally those in the posterior triangle of the neck. The swelling thus occasioned alters very materially the natural configuration of the neck. General and local medicinal treatment may bring about considerable diminution in the size of the glands, but the enlargement rarely disappears completely, and when situated near the angle of the jaw may still be easily detected. The patient is probably unaware of or indifferent to their presence, as they are not visibly enlarged. As soon, however, as the general health becomes impaired, the glands again become prominent.

This alteration in size may extend over many months, and with each reaction there is a development of cicatricial

tissue which leads to the intimate attachment of the glands to the structures around them. The anatomical character of the majority of the glands is lost, and the whole or the greater part of the gland is replaced by caseous matter. The skin is not affected, but may be adherent to the underlying fascia. The disease in this type runs essentially a slow course, but, as has been already stated, is subject to variations. There comes a time, however, when the glands remain enlarged and neither local nor general treatment makes any impression on them. It is then that surgical measures are entertained, though reluctantly, as the disfigurement cannot otherwise be removed, and the cicatrices from the usually employed methods seem to be the only alternative to the unsightly marks which follow the breaking down of the glands and the consequent involvement of the skin.

In the second type, softening of the caseous matter will be found to have taken place. This is determined by the presence of tension or fluctuation. In cases of doubt as to the state of the gland, a fine grooved needle may be introduced. In one or two cases this has indicated the presence of pus which otherwise could not have been detected, and enabled the operator to adopt prompt surgical measures.

The glands in front of the sterno-mastoid muscles are usually the first to show signs of breaking down. If they have been previously affected the pus-formation may be very rapid and the surrounding tissues become involved. This fact is of considerable importance, and one which perhaps has not been fully grasped by many who are concerned in the treatment of tuberculous glands. Measures which may have been effectual in reducing the enlargement on one or two occasions may subsequently fail; and if an abscess forms and the skin becomes damaged, reflections will probably be made on the treatment.

My experience of this type and of the difficulties they present leads me to strongly urge their removal while in the quiescent stage.

In the third type, the area involved is a very large one.

Not only are the glands indicated in the preceding types affected, but those beneath the chin and above the clavicles are here also involved, and generally on both sides. Although I have removed glands from these areas to the number of seventy on one side through an incision in the hairy scalp, I do not advocate this operation in such cases. It should be limited to the removal of those glands lying beneath and behind the sterno-mastoid muscles.

Operation ; preparation of the parts.—The utmost care should be taken to render the parts aseptic. The head is thoroughly cleansed beforehand and the scalp shaved over the spot at which the incision is to be made and for about two inches above this. It is not necessary to sacrifice a large quantity of the hair, and in the case of one young woman what was removed was afterwards utilised to cover the cicatrix until the new growth was sufficient for that purpose. The shaven part of the scalp, the ear, the face and neck, being the parts with which the hands of the operator are brought into contact, are treated with spirit of soap and then with methylated ether, and the area of operation is then limited by towels held in position by pressure forceps.

The incision is made in and near the edge of the hairy scalp from a point near the base of the mastoid to the nape of the neck. By thus following the outline of the hairy scalp the wound has its convexity downwards, and the centre of it is so brought nearer to the affected glands. The skin along the lower edge of the wound is carefully freed and drawn downwards until the glands in the upper part of the posterior triangle come into view. There is no danger to structures of any importance at this stage, the lesser occipital nerve being defined and in some cases separated without difficulty.

The spinal accessory nerve is easily found and detached from the glands. Bleeding from one or two small points is controlled by pressure forceps. The glands nearest the wound are now seized with pressure forceps, and their separation from the fascia is easily effected by means of an ordinary pointed elbow scissors, which is introduced closed

and then opened; this is repeated at various points around the glands.

The posterior border of the sterno-mastoid muscle is next defined, and with a little careful dissection it can be raised from the glands below it without damaging its fibres. In the removal of the glands beneath the sterno-mastoid muscle the internal jugular vein stands in danger of being injured, either by undue traction being made on it or by puncture with an instrument. If a deliberate dissection is made in order to expose the vein at once, these accidents may be avoided. This is effected by the removal of one or two glands from the centre of the mass. When this is accomplished the vein is then pushed aside, and the glands above and below the opening thus made can be easily and satisfactorily dealt with.

Isolated glands just above the clavicle, and the chain of glands along the posterior border of the sterno-mastoid muscle, are reached through the same incision.

The two forefingers are now introduced into the wound beneath the sterno-mastoid muscle, to detach the deep cervical fascia from the glands near the angle of the jaw. A pair of strong dressing forceps also helps to complete the separation of the fascia. Should a gland have undergone softening, an incision is made into it and its contents scooped out, the capsule being removed later. This procedure prevents infection of the tissues and affords better access to the other glands.

The value of a good light at this stage is inestimable. On raising the sterno-mastoid muscle with broad retractors a good view is obtained of the parts below and in front. The posterior belly of the diagastric muscle should be defined, as in separating glands beneath the plane of this muscle the hypoglossal nerve and the occipital artery may be injured; this, however, can be avoided by the exercise of a little care. But, the field of operation lies above the digastric muscle, and here the facial vein calls for some attention. Hemorrhage from an injury to it is usually free, and owing to the depth of the wound is not easily arrested.

In only two instances in twenty operations was it damaged. It may be stated here that as a rule bleeding is trifling, and in many of the operations no vessel whatever was ligatured.

A careful examination is made by the finger for enlarged glands, in or near the lower part of the parotid gland, below the chin, and in the angle between the sterno-mastoid muscle and the greater cornu of the hyoid bone. In the latter situation, and close to the internal jugular vein, a gland of considerable size is frequently found. These glands are removed in the manner already detailed. Should those situated beneath the chin have undergone softening, they are removed through a small incision made directly over them. Retractors are again used to allow of a careful inspection of the wound. With a sharp knife or scissors and dissecting forceps, any part of the capsule of the gland which has been left is dissected away, and as the internal jugular vein, the carotid arteries, the facial vein, the occipital artery, and the spinal, accessory, and hypoglossal nerves are in view, this part of the operation is not attended by much difficulty or danger. A large drainage-tube is inserted and the wound closed with silkworm sutures. The dressing is applied in the usual way. The drainage-tube is withdrawn about the end of the first week. Occasionally some fulness appears over the sterno-mastoid muscle after the wound has healed, but it is readily dispersed by a gentle massage. The pressure of an elastic bandage, which need only be worn at night, also assists in restoring to the neck its natural form.

In a paper on the surgical treatment of tuberculous glands in the neck which appeared in a recent number of the 'Journal of the American Medical Association,' reference was made to an operation by Dallinger by which the glands were removed through the hairy scalp. No details of the method were given, so it is impossible to state how far it differs from the operation I have practised. I may add that my first patient was operated on 4½ years ago.

(Read at the Provincial Meeting, Brighton, June the 20th, 1903.)

Discussion.

The Chairman (**Dr. Wayland Chaffey**) (Brighton) thought there were several members present who had had considerable experience in the removal of large masses of glands from the neck, though in and around Brighton one did not see such large masses in that situation as were seen in other parts of the country. He had seen very large masses of cervical glands whilst he was at children's hospitals in London. Mr. Morgan, who was attached to The Alexandra Hospital, would no doubt speak on the subject.

Mr. George Morgan (Brighton) thought the paper was interesting, but that it would require very careful technique to prevent suppuration at such a depth. Moreover, the drainage there could not be very good. The glands were not seen sufficiently early at The Alexandra Hospital; the children were generally brought when the skin was giving way, at which stage he supposed Mr. Parry would not carry out his method, especially if there was an abscess under the skin. Usually he only had a small scar resulting from his operations on those glands. If care were exercised there was few relapses.

Mr. R. Clement Lucas said he had listened with the greatest possible interest to Mr. Parry's remarks, and it seemed that in a certain number of cases the author could avoid a scar in the neck and produce very good results. He thought the number of such cases must be limited. He had been in the habit of teaching that to attempt to remove from the neck tuberculous glands which had undergone suppuration was one of the most difficult operations in surgery. One often found that the adhesions to nerves, muscles and vessels were as strong as in cancer. Those cases would be unsuitable for the procedure just described. The plan of Mr. Parry showed a great advance in a certain limited number of cases. He asked whether Mr. Parry was able to get out of that high incision the glands immediately above the clavicle. When the post-sterno-mastoid glands were enlarged they generally extended in a chain right down to the glands on the top of the clavicle. He also did not understand how Mr. Parry secured the facial vein in those cases in which he damaged it. He asked if the sub-maxillary glands were pushed under the sterno-mastoid, or were they moved over the surface of the sterno-mastoid?

Mr. A. H. Tubby congratulated Mr. Parry on his new procedure. Such a method was of interest because it was new. He could imagine some conditions in which the operation would be advisable. He did not gather how far into the hairy scalp the incision was made; whether it was carried sufficiently high to be quite invisible and if there were a due growth of hair afterwards. He would like also to hear how long the operation

took compared with that occupied by direct incision over the site of the trouble. He could not help feeling, that though the method was good cosmetically it yet introduced a considerable amount of danger into the case. It was very difficult to avoid the intricate and complicated structures in the neck in a deep wound. Also he wished to ask as to the incidence or absence of suppuration. He simply asked for information, as he had not performed that particular operation, and it might be that the cases did not present those possibilities which one would expect.

Mr. R. H. Parry (Glasgow), in reply, said he did not find much trouble in ligaturing the facial vein. The structures could be pushed into the wound, which lay near the sterno-mastoid. He was able to pull the internal jugular vein into the wound, and then secure the facial. The hæmorrhage was fairly free, but by putting his finger on the vein and drawing it forward he was able to manage all right. He did not now expect that accident, in the light of greater experience. The position of the cicatrix was half an inch from the edge of the scalp. People had been so pleased with the results of that operation that they would not have the usual one done. With regard to suppuration the drainage was excellent; the tube was at the back, and the patient lay on his back. He had very slight suppuration in two cases, and that he believed to be due to carelessness on the part of his house-surgeon. He had not seen it in private cases. He took great care to cleanse the scalp and its neighbourhood, and he had only had trouble in two cases out of twenty. He believed his operation was easier than the old one; it was easier to separate the glands from the vessels from below than from above. The attachment of the deep cervical fascia was very intimate, but working from below was surprisingly easy. He got over the difficulty in the first stage of the operation. He had removed a fairly big mass of glands comfortably in half an hour, without tying a vessel, and he could be certain there was not a gland left. In reply to Mr. Clement Lucas's question, the glands were carried under the sterno-mastoid.

64. A CASE OF SYPHILITIC NEPHRITIS IN AN INFANT AGED FIVE MONTHS.

By GEORGE CARPENTER, M.D.

Charles B—, aged 5 months, was admitted into the North-Eastern Hospital for Children on May the 29th,

1903. His mother attended Shadwell Hospital six years previously for a venereal complaint, and his father was treated as an out-patient at St. Bartholomew's Hospital about the same time for a disease for which he took a prolonged course of pills. The first child was prematurely born at 6½ months; it lived 2 months. The second was a full-term child and is still living; the third was also full-term and is living; and the fourth, the patient, was an 8-months child.

At birth it was a small but apparently healthy infant. It was breast-fed. At two months old it had "snuffles" and "congestion of the lungs," and began to waste. Next a red rash appeared on its buttocks and its legs and feet. This faded and then disappeared. The child was always pale, and its face was noticed to be "puffy," more so at one time than another.

On admission his weight was 9 lbs. 7 oz.; the motions were green and slimy, and the temperature was 97·8° F. He was a small, anæmic infant, with scanty hair on the front half of the scalp; the eyebrows and eyelashes were also thin. The face was puffy, especially below the eyes, and the temporal veins stood out prominently. "Snuffles" was a marked feature. He had a copper-coloured patch of eruption on the chin, and copper-coloured macules were scattered about the buttocks. There was also copper-coloured redness in the flexures of both knees, which extended on the left side to the calf. The palms were glistening. The heart, lungs, liver, spleen, and abdomen were healthy. The eyes and fundi oculorum were normal. The urine was of specific gravity 1006, the reaction was acid, and it contained ¼th of albumen. He was ordered Hydrargyrum cum Cretâ, one grain, thrice daily.

April the 28th.—The quantity of urine passed in the twenty-four hours was 3 ounces. The specific gravity was 1008, the reaction acid, urates were deposited, and the albumen amounted to one third.

30th.—The rash is fading. The quantity of urine passed in the twenty-four hours was 2½ ounces. The

specific gravity was 1006, the reaction acid, urates were deposited, and the albumen amounted to one third.

May the 2nd.—To-day the face is very "puffy," and the legs pit on pressure. The quantity of urine passed was 3 ounces. The specific gravity was 1010, the reaction acid, urates were deposited, and the albumen was one third.

6th.—The urine was of specific gravity 1006. The urea was estimated at $1\frac{2}{3}$rds of a grain to the ounce. Albumen amounted to five twelfths. On centrifuging the urine an appreciable quantity of detritus appeared in the tube, which consisted of: (1), numerous hyaline casts, both large and small; (2), similar casts with a few epithelial cells and red blood-discs upon them; (3), definite epithelial casts; (4), red blood-discs; (5), detritus of renal epithelium, the cells appearing singly and in clumps; (6), granular casts, a few; and (7), urates of sodium and ammonium crystals. There were really quite a number of red blood-discs which were swollen, more than a sprinkling. The microscopical characteristics were therefore those of acute catarrhal nephritis.

8th.—There has been a loss of weight, $\frac{3}{4}$ths of a lb., since his admission into the hospital. The temperature keeps subnormal. The quantity of urine passed in the twenty-four hours is slightly more, viz. $4\frac{1}{2}$ ounces. The specific gravity is 1008, it is acid, urea amounts to $1\frac{1}{3}$rd grains to the ounce, and albumen is one fifth. Renal casts and urates are present. The rash on the chin is fading. There is marked pitting of the legs and slight pitting of the backs of the hands on examination.

10th.—The quantity of urine passed in the twenty-four hours amounted to 5 ounces. Urea was $1\frac{2}{3}$rds of a grain to the ounce, and albumen was one fifth.

12th. Four ounces of urine were passed in the twenty-four hours, and the albumen was one fifth.

14th. Seven ounces of urine were passed, and albumen amounted to one sixth.

16th.—Five ounces of urine were passed, and albumen

amounted to one sixth. The temperature rose to-day to 99·4° F.; it had been subnormal since his admission.

18th.—The face is more swollen, especially in the morning. There is much dropsy of the legs, extending above the knees to the buttocks. The child is wasting, and is failing in strength. The eruption has disappeared. The buttocks are superficially ulcerated.

20th.—Six ounces of urine were passed of specific gravity 1002, urea 1⅓rd of a grain to the ounce, and albumen one eighth. The temperature was normal. The general œdema had increased.

21st.—The urine was swarming with organisms, and casts were very few. One or two epithelial casts were seen, and some hyaline and waxy casts. There were no red blood-corpuscles. Round-cells were seen about the size of or a trifle larger than white corpuscles. The specific gravity was 1001, the reaction acid, urea was 2 grains to the ounce, and albumen was barely one sixth.

23rd.—He is now passing 9 ounces of urine of specific gravity 1002, and acid reaction. Albumen amounts to one seventh. He is much weaker, and the œdema is increasing.

25th.—Six and a half ounces of urine were passed, and albumen was one sixth. The temperature now varies between 97° F. and 98° F., and the child is growing perceptibly weaker.

29th.—He sank and died in the early morning.

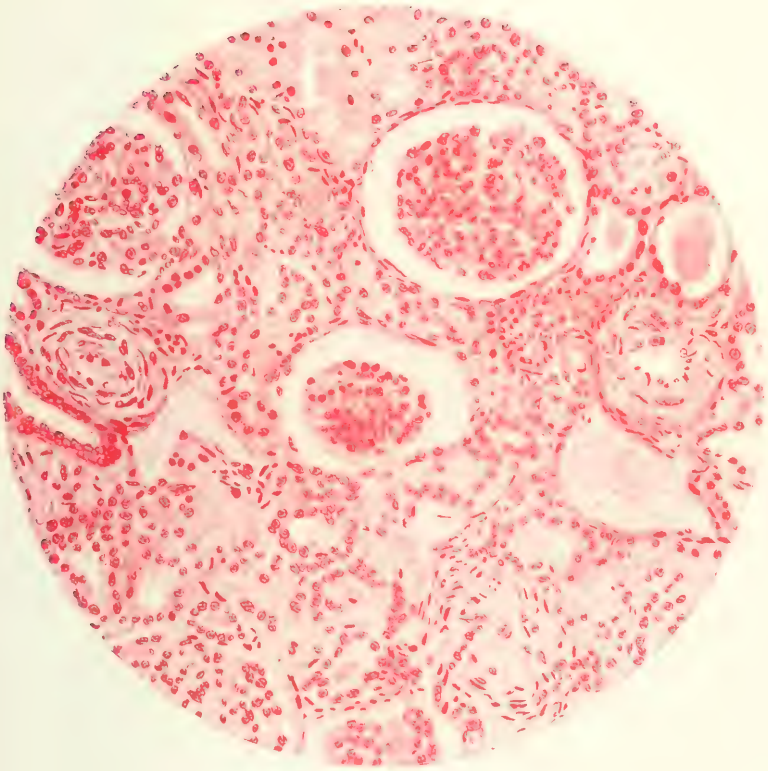
Post-mortem examination.—The heart and lungs were normal. The spleen was normal; the liver was toughened (?). The kidneys were lobulated, and the capsule readily stripped; coarse changes were not obvious.

The microscopical report is as follows:

The kidney is in a condition of catarrhal nephritis. Casts are visible here and there in the tubules. The cortical renal tubules are dilated. There is an excess of round-cells in the glomeruli, and fibrillary thickening of the glomerular capsules. The catarrhal changes are patchy in distribution, some areas of the kidney being apparently quite healthy. There are not any interstitial changes.

The small arteries in the neighbourhood of the glomeruli are thickened, and their lumens are greatly diminished (*vide* Plate III). This thickening appears to be due to mesarteritis rather more than to endarteritis, but in some situations the inner coat shares and the arteries are obliterated, or nearly so. The endothelium of the glomeruli seems to be thickened also; at least, that is my opinion on comparing the specimen with a healthy infantile kidney. I think the structural alterations in the blood-vessels may fairly be held responsible for the anuria which was a prominent feature in the case. I have compared many specimens of catarrhal nephritis in non-syphilitic children with the one in question, and the arteries were not thickened in these. I am inclined to look upon these arterial changes as syphilitic.

The next point is, can catarrhal nephritis arise from the action of the syphilitic poison? My reply to that is, Yes, undoubtedly; and that on clinical observation backed by pathological finding. Here is the proof. A weakly-looking, "snuffling" female infant, aged five weeks, came under my care at the Evelina Hospital on November the 7th, 1889. A rash appeared when it was two weeks old. When I saw the child there was desquamation about the palms, the soles, the belly, the ears, and elsewhere about the body. The lips were superficially ulcerated. There was shallow ulceration from the anus upwards between the gluteal folds. At the root of the nose there was a yellow scab, and slight yellow crusting here and there on the ears. On the buttocks there were a number of red papules with crater-like central depressions. A week later the back of the left hand became puffy. The following week there was a deep ulcer in the natal fissure with a yellow unhealthy base. Three weeks from the first attendance both eyelids were œdematous, white, and waxy-looking, and there was marked dropsy of the feet, legs, hands, and forearms. There was a ring of albumen with cold nitric acid $\frac{1}{16}$th of an inch thick. Microscopically, in a gravity specimen, there were hyaline, and epithelial, and a few blood-casts, but no separate red blood-discs. The child



Dr. George Carpenter's case of Tubular Nephritis with thickened Arteries, in a Syphilitic Infant aged 5 months.

died soon after this. As in the case previously described, Hydrargyrum cum Cretâ, one grain, was given thrice daily.

Post-mortem examination.—There was consolidation of both lower lobes of the lungs. The kidneys, which were quite natural-looking, were lobulated; there was doubtful injection, in places, of the interglobular vessels. I have drawn attention to this case in my book on 'Syphilis in

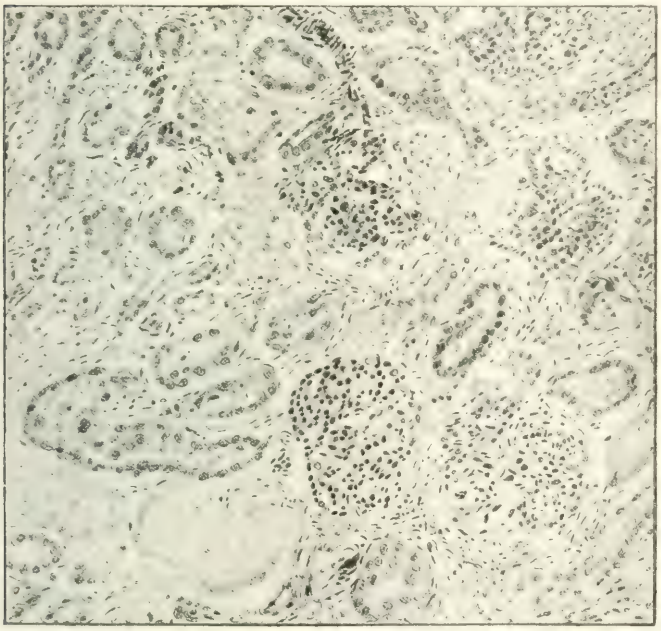


Fig. 28.—Microscopic section of the kidney of a syphilitic infant, aged 5 weeks, showing interstitial and catarrhal nephritis.

Children,'* and a microscopical drawing of the kidney will be found on page 56. The original of this I now pass round for inspection. The lungs showed grey hepatisation; the alveoli were more or less blocked with round-cells. There was fibrosis of the vessel walls.

The kidneys showed intense glomerulitis, and the

* London, 1901: Baillière, Tindall, and Cox.

glomerular capsules were thickened by the addition of fibrous tissue; there was also interstitial fibrosis (see Fig. 28, page 291). The vessels seen in transverse section have their walls thickened by the addition of fibrous tissue (mesarteritis), and there is an indication of new vessel-formation in the young fibrous tissue which is developing in the stroma. In one section a round-celled infiltration, measuring 0·033 by 0·0166 mm., was large enough to constitute a tiny granuloma. The tubules were in a state of catarrhal nephritis. As in the other case, the lesions were patchy in distribution.

Here, then, there is evidence of catarrhal nephritis in an incontestable syphilitic kidney, and the fact that syphilis can and does produce catarrhal nephritis is obvious.

There is no reason, therefore, why a point should be strained to prove that the case of catarrhal nephritis I now place on record is not due to syphilis because there are no interstitial changes, and that it must on that account be owing to some other undetermined cause; on the contrary, quite the other way. I purposely ignore the condition of the small arteries, which I take to be a syphilitic change.

I am convinced from this case that congenital syphilis is capable of producing catarrhal nephritis as well as interstitial nephritis, and there is no valid reason why one form should not occur independently of the other.

These, unfortunately, are the only pathological findings of syphilitic kidney disease which I have had under my clinical observation. I have had from time to time, though very infrequently, several cases of catarrhal nephritis in syphilitic infants, which have unfortunately been lost sight of; but, as is conclusively shown above, the finding of catarrhal products in the urine does not prove the absence of interstitial changes in the kidney.

The practical lesson to be learned from these clinical observations is that congenital syphilis in some instances produces a nephritis which is indistinguishable clinically

from acute catarrhal nephritis by reason of its symptoms and its urinary deposits, and that interstitial kidney changes may or may not be present in such cases. Further, congenital syphilis plays a more important part in the production of renal disease in the young than has hitherto been conceded. Dr. Henry Ashby in 1901 brought to the notice of the Society a case of nephritis in a four-weeks-old infant who was very anasarcous, and died in convulsions soon after admission into the hospital. The urine was not obtained for examination.* It was thought to be possibly of intra-uterine origin. The post-mortem changes were catarrhal and interstitial, and there was fibrosis of the glomerular capsules. The small arteries appeared thickened. There was no history of syphilis, but the mother had had one stillbirth. The changes in the kidney at the time reminded me of my example of catarrhal and interstitial nephritis in a five-weeks-old infant who was syphilitic, and I suggested on that finding that the condition was of syphilitic origin.

The quantity of urine passed daily by the infant was very low indeed. In a series of observations which I made on the urine of a newly born infant some time ago, and the results of which I brought to the notice of the Society,† the average quantity for the twenty-four hours was $19\frac{1}{2}$ ounces. At this point I should like to record the fact that the child on whom these observations were made is now quite well and strong, and free from disease. The urea in that case was very small, from $\frac{1}{4}$th to $\frac{1}{6}$ of a grain per fluid ounce, the greatest daily quantity being $4\frac{1}{2}$ grains and the smallest $2\frac{4}{5}$ths grains.

In comparison with this, the urea obtained from the case of nephritis does not seem unduly low, though the normal has been estimated at a somewhat higher figure than this

* "A Case of Nephritis in a Newly Born Infant," 'Reports of The Society for the Study of Disease in Children,' vol. i, pp. 129—131.

† "Notes on the Urine of a Newly Born Infant," by George Carpenter, M.D., 'Reports of The Society for the Study of Disease in Children,' vol. i, p. 160.

by some foreign observers. The quantity of urea passed per ounce would accord with observations I have made on healthy children about the same age, but in these I have no record of the amount of urine passed in the twenty-four hours. If the quantity of fluid passed by them be standardised by the test infant, the amount of urea, a trifle over 8 grains, excreted by the syphilitic infant in the twenty-four hours was subnormal.

Considering the small daily quantity of urine passed, the specific gravity was unduly low; it accords, however, with that of the test infant, but in this case the total daily amount was much higher.

(*Read at the Provincial Meeting, Brighton, June the 20th,* 1903.)

Discussion.

The Chairman (**Dr. Wayland Chaffey**) (Brighton) said Dr. Carpenter's paper showed great research, and could not be prepared in a few weeks, or even months. The drawings and specimens also were very nicely prepared, and the Society was much indebted to the author.

Dr. G. A. Sutherland said the subject was one which the Society had been concentrating its attention upon for some years past. Dr. Guthrie, a few years ago, wrote and read a paper on chronic interstitial nephritis in children, and supported the view that most of such cases were due to syphilis. During the past winter Mr. Thomson Walker and he (Dr. Sutherland) read a paper on two cases of congenital nephritis in infants, and Dr. Carpenter had brought up an interesting series on the same lines. His (Dr. Carpenter's) chief point was that in the case of catarrhal nephritis he found evidences of syphilis in the child, and he was inclined to state rather positively that the nephritis was dependent upon syphilis. It was open to discussion whether it should be put into that category. He was inclined to agree with Dr. Carpenter in regard to the specimens he had brought, but he thought the evidence was not so conclusive as one would like to have it in settling such a point. The changes shown in the specimen were largely those of catarrhal nephritis, and there was considerable thickening of the arteries in addition. In one of the cases under the care of Mr. Walker and himself there was a marked thickening of the cerebral arteries, in addition to the nephritis. In those cases one would look for, and expect to find, definite arterial changes associated with syphilis. Dr.

Carpenter's cases were different from the ones Dr. Sutherland had seen, in that the patients had, during life, the definite signs of nephritis present, œdema, and the presence of tube casts in the urine. He supposed that in most of the cases the changes were chronic interstitial, because there were no signs of catarrhal nephritis during life, and in most of the cases the disease was discovered accidentally post mortem. The cases he had brought forward were practically pure interstitial nephritis, there being so much exudation that the tubes were almost blocked, and one could not make out catarrh, except that which was secondary to pressure from the exudation. He asked whether Dr. Carpenter had examined, in his case, the condition of the cerebral arteries, because if that were not done the condition was liable to be overlooked. In one of the cases he had with Mr. Walker one of the diagnoses suggested during life was syphilitic endarteritis, consequently the arteries at the base of the brain were very carefully examined. It was the only case in which he had found that condition present.

Dr. George Carpenter, in reply, said he did not examine the brain, as he was not allowed to do so. There were, however, no signs of cerebral disease in his case, but in the case published by Dr. Sutherland and Mr. Thomson Walker there was, if his memory served him correctly, evidence of cerebral disease during life. His first experience of kidney disease in congenital syphilis had occurred as far back as the year 1889, and he had recorded his own observations on this subject, in his book on 'Syphilis in Children,' which he feared had been overlooked. He was not moved by Dr. Sutherland's criticism as to the diagnosis: indeed, he was under the impression that Dr. Sutherland had arrived at the same conclusion in his cases by a similar process of deduction of which he now complained. The cases he had observed during life in syphilitic infants had given clinical evidence of catarrhal nephritis, and after death he had found in one case a mixed catarrhal and interstitial nephritis and in the other catarrhal nephritis. His experience had been mainly clinical and it was attended by pathological findings in the two experiences he was enabled to relate. He had not met with in the post-mortem room, by accident as it were, such cases as Dr. Sutherland and Mr. Thomson Walker had placed on record. On the other hand he had examined the urine of a number of cases of congenital syphilitic infants and he had not found evidence of nephritis, catarrhal or otherwise. Albuminuria in infants must not be looked upon as indicating nephritis apart from definite physical signs and the finding of renal inflammatory products in the urine.

65. ON SPLENOMEGALY IN INFANTS AND YOUNG CHILDREN.

By GEORGE CARPENTER, M.D.

Enlargement of the spleen is not an uncommon manifestation in early childhood, and it is frequently found enlarged in infancy.

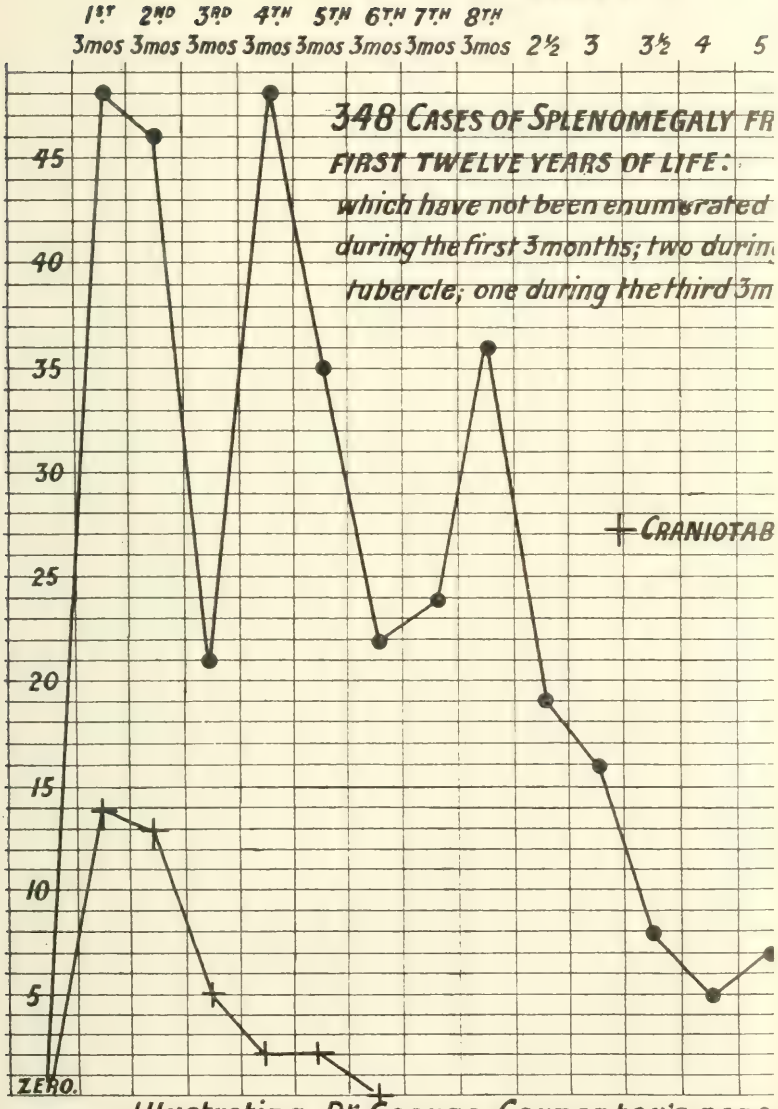
The clinical material which has passed through my hands during the last few years at the Evelina and the North-Eastern Hospitals for Children comprises 348 cases in which the spleen was enlarged, as determined by palpation, from all causes during the first twelve years of life, cases such as are commonly found in out-patient hospital practice, and which have not been selected.

For convenience of description I have arranged the cases in charts (*vide* Chart I).

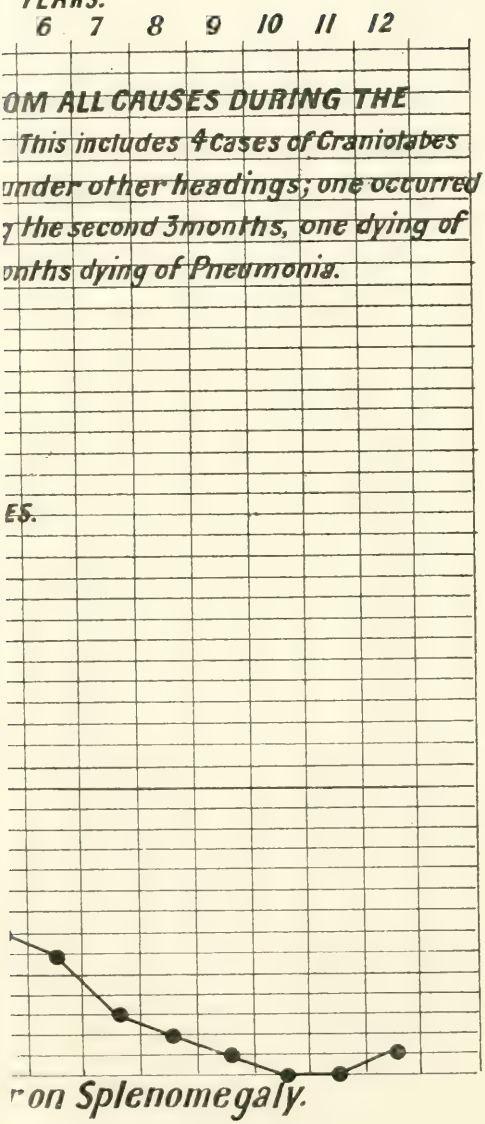
In this chart each horizontal line denotes one case, and each period of time which has been selected for examination comprises three months, and is bounded by a vertical line; thus during the first two years of life eight three-monthly periods will be noted. After the first two years of life the periods of time have been extended to six-monthly intervals for the following two years, and to yearly intervals after that time.

A study of this chart shows that during the first six months of life cases of splenomegaly are very frequent indeed. By the ninth month there is a great diminution, and the number has decreased by more than half. This diminution, however, is followed within the next three months by a very rapid rise up to the first year, the number increasing to as many as that met with during the first three months of life. A fall in numbers next occurs, and when the eighteenth month is reached cases are about as numerous as at the ninth month of life. Then there is a steady rise in the numbers, which about equal that of the fifteenth month, and from that period

CHART I.

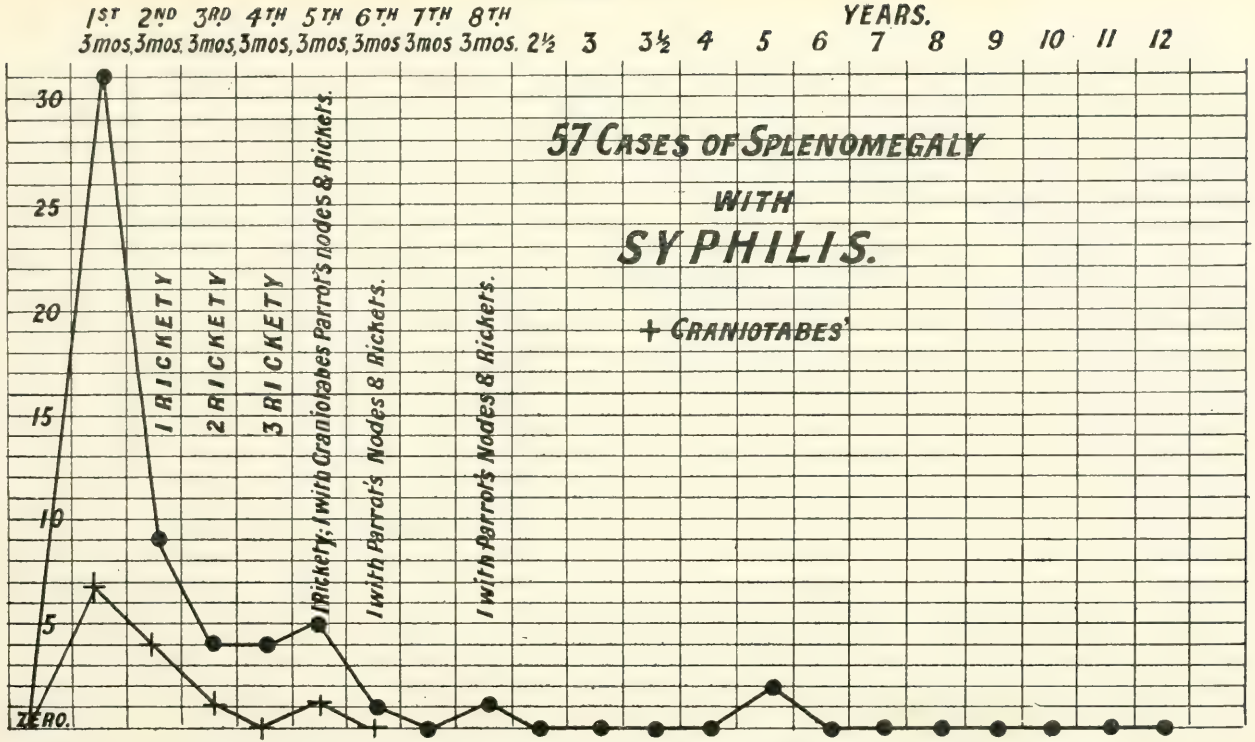


June 1903. Illustrating Dr George Carpenter's pape


YEARS.
6 7 8 9 10 11 12
OM ALL CAUSES DURING THE
This includes 4 cases of Craniotabes
under other headings; one occurred
the second 3 months, one dying of
onths dying of Pneumonia.
ES.

r on Splenomegaly.

CHART 2.



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to the fourth year there is a steady fall, and by that time cases are becoming uncommon.

In this chart three large pinnacles of splenic enlargements are depicted: a lofty pinnacle during the first three months, which maintains its height for the following three months; an equally high pinnacle at the twelfth month; and a well-marked pinnacle, but of less altitude, at the second year.

Splenic enlargements are very numerous, therefore, during the first two years of life, amounting to something like seven ninths of the total number of my cases.

Chart II comprises fifty-seven cases of undoubted syphilis associated with enlargement of the spleen. The cases have been arranged on a similar plan to that adopted in Chart I.

It will be seen that the top of the syphilitic pinnacle is within the first three months of life, and that by the ninth month the number of cases has very greatly diminished, but remains more or less steady for the following six months, and that from the eighteenth month onwards undoubted cases of syphilis with splenomegaly are quite uncommon. It is interesting to note that four of my cases in which the spleen was enlarged attended the hospital for cauliflower-like condylomata ani, viz. two at five years of age, one at thirteen months old, and one at fifteen months old, without other evidence of syphilis.

In the vertical columns the number of these cases that were rickety also is indicated; they amount to ten instances, and out of fifteen cases nine were rickety.

There were three cases of cranial osteophytes (Parrot's nodes), all of these cases being rickety likewise, and in one case craniotabes was associated. The number of cases with craniotabes in this series is also indicated. It will be noted that craniotabes is most usually encountered during the first six months of life. In Chart I all the cases which had craniotabes are denoted by a double line, and the same feature is prominent, viz. that craniotabes is most frequently met with during the first six months of life.

In my experience splenic enlargements in children up to six months of age may be estimated to occur in about 40 per cent. of indisputable syphilitic infants.*

Passing now to Chart III, there are there represented sixty cases with enlargement of the spleen and chronic "snuffles," and over one third of these cases were associated with craniotabes. Here the same feature is prominent, that craniotabes is most common during the first six months of life. Of these sixty cases twenty-three were rickety, the majority being decidedly rickety, and of these rickety children two were very rickety, and four among them had Parrot's nodes. There were also two cases of Parrot's nodes that were not rickety.

A doubtful sign of congenital syphilis when taken alone is perhaps of no great value, but when two or more doubtful signs occur in conjunction the balance of evidence is then strongly in favour of a positive diagnosis.

Chart IV concerns thirty cases of splenomegaly, wherein syphilis could not be excluded by reason of the family history or of some doubtful personal factor.

Of these cases seventeen showed some signs of rickets, and the bulk of them were definitely rickety, and one was very rickety. Two of the rickety cases had Parrot's nodes. There was but one case of craniotabes among them, and that was a very late example, viz. during the twenty-first month of life.

If the obvious, the probable, and the possible cases of syphilis be added together the total amounts to but 147 cases, 201 short of the total number of enlarged spleens.

I propose next to deal with several physical signs which are frequently associated with splenic enlargements, and are therefore of more than passing interest, and to which I have, in times gone by, devoted some study; I refer to *craniotabes, cranial osteophytes,* and *chronic "snuffles."*

It will be noticed that I have purposely in the charts not set any specific value to the two former signs, so that the issue may not be confused; but attention has been

* 'Syphilis in Children,' by George Carpenter, see p. 50.

CHART 3.

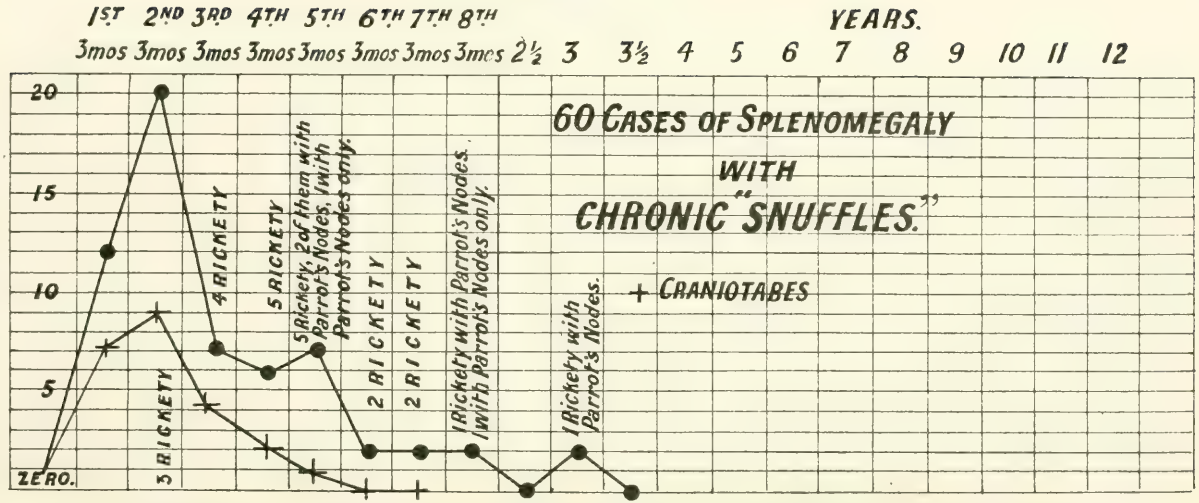

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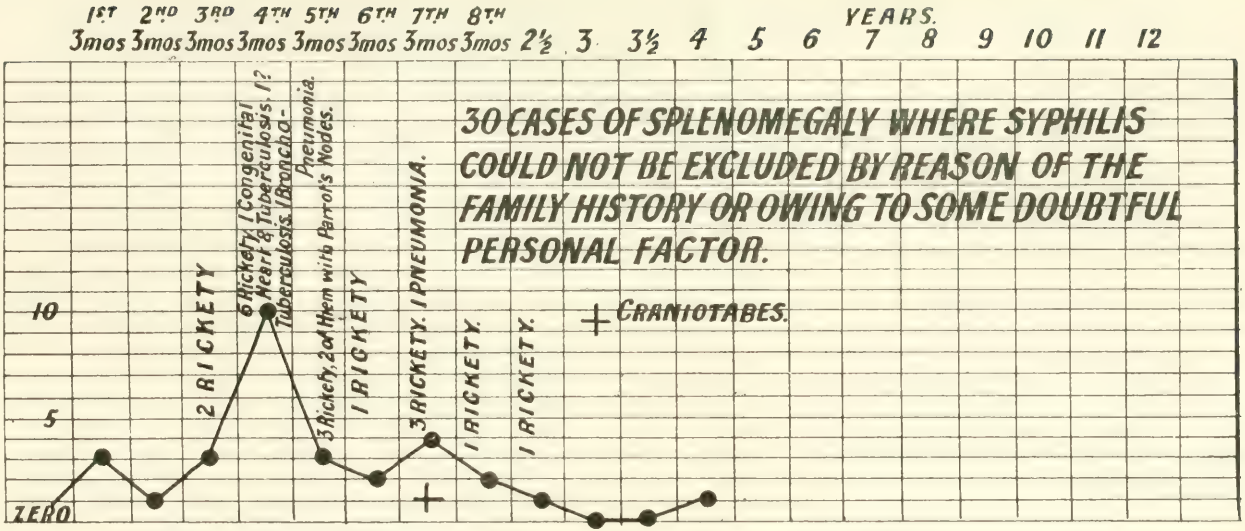
CHART 4.


1ST 2ND 3RD 4TH 5TH 6TH 7TH 8TH
3mos 3mos 3mos 3mos 3mos 3mos 3mos 3mos
YEARS.
2½ 3 3½ 4 5 6 7 8 9 10 11 12
30 CASES OF SPLENOMEGALY WHERE SYPHILIS COULD NOT BE EXCLUDED BY REASON OF THE FAMILY HISTORY OR OWING TO SOME DOUBTFUL PERSONAL FACTOR.
10
5
ZERO
2 RICKETY
6 Rickety, 1 Congenital Heart & Tuberculosis. 1? Tuberculosis. 1 Broncha-Pneumonia.
3 Rickety, 2 of them with Parrot's Nodes.
1 RICKETY
3 RICKETY. 1 PNEUMONIA.
1 RICKETY.
1 RICKETY.
+ CRANIOTABES.

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drawn to these conditions, and the number of cases suffering from them has been duly noted in the charts.

In regard to craniotabes, the remarks which I am now about to make in reference to the ætiology of this manifestation are the outcome of a study of 238 cases of that condition.* †

These cases did not comprise instances of thinning of the skull bones in the neighbourhood of the cranial sutures and the fontanelles, but were examples of thinning of the cranial vault away from the sutures. I have already pointed out elsewhere that the parietal bones are those most commonly involved, and the frontal bones rarely suffer, and then only in association with other cranial bones. The most prolific period of the condition is during the first four months of life, and it is very uncommon from the ninth month onwards, and by the sixteenth month it is a rarity. During the first six months, or what I will term the active syphilitic period of life, it is very common, and during the active rickety period it is quite uncommon, and when the children are most rickety that is the least likely time to find it. If a line of cases of craniotabes be drawn in chart form step by step with a line which denotes the syphilis cases among them, it will be noted that the syphilitic line follows the craniotabic with great fidelity. A common combination with craniotabes is chronic "snuffles." Craniotabes and chronic "snuffles" may be the only indications of the syphilitic taint, and I have on numerous occasions proved the truth of the suspicion by an examination of other children of the family, or of the parents, or by being previously acquainted with the fact that one or other of the parents was syphilitic.

Infants wasted from dietetic errors are not, in my experience, craniotabic; indeed, it is surprising how hard the skulls are of some of these infants.

There is one other point; craniotabes sometimes readily

* 'Syphilis in Children,' by George Carpenter, pp. 74—78.

† 'Reports of The Society for the Study of Disease in Children.' *Vide* fig. 21, p. 215, vol. iii.

responds to mercurial treatment, at others that is not the case. Craniotabes may recover, and rickets, if associated with it, may persist and increase. The presence of craniotabes in an infant should in my opinion excite a strong suspicion of syphilis.

Cranial osteophytes * (Parrot's nodes) are not infrequently associated with an enlargement of the spleen—about half of the cases in my experience. In a series of observations which I made on this condition I found 57·5 per cent. of the cases were undoubtedly syphilitic.† Seeing the difficulty of detecting syphilis at the period of life at which these nodes are usually to be found, so high a percentage of syphilitic cases is *very* striking. Rickets is not an essential factor in the production of Parrot's nodes, for these may be occasionally produced when rickets is absent or but slightly marked. The majority of patients, however, are more or less rickety, and some are *very* rickety. The condition is usually encountered during the first two years of life, being very rare under six months of age. I found it associated with chronic "snuffles" in a quarter of the cases. In three instances out of forty I observed craniotabes in association with Parrot's nodes. Occasionally the condition is combined with periosteal nodes elsewhere, viz. in the long bones or in the phalanges. Antisyphilitic treatment, in my experience, has had no effect upon them, with one or two exceptions I can call to mind, and the peculiarity of the skull when once contracted may be detected with more or less ease at puberty, though time tends to tone down the condition.

I am in favour of the view that Parrot's nodes is a syphilitic manifestation, and the condition always excites in my mind the strongest suspicion of that complaint.

Of eleven cases dealt with here, three will be found in Chart II in syphilitic children, one of them being craniotabic. Six will be found in Chart III associated with

* *Vide* Postscript, 'Reports of The Society for the Study of Disease in Children,' vol. iii, p. 221.

† 'Syphilis in Children,' by George Carpenter, pp. 70—74.

chronic "snuffles" and splenic enlargements, and two will be found in Chart IV, which deals with doubtful cases of syphilis.

Chronic "snuffles" is a very important condition in infancy. It is usually admitted that infants are liable to coryza; indeed, many infants are introduced into the world with a non-specific cold in the head. Such troubles are of short duration usually, though they may recur. Chronic "snuffles," or a chronic cold in the head, is quite another matter. It is the *long* duration that distinguishes the complaint from the simple affection. In a certain proportion of nurselings it is possible in such cases to feel the posterior nares and exclude congenital adenoids, but in others it is impossible to make a digital examination. In older children the subject of chronic "snuffles" adenoids are not infrequently to be felt, but it is equally true that they are not infrequently absent. I am under the impression that chronic naso-pharyngeal catarrh is conducive to adenoids, and certainly the detection of congenital adenoids does not exclude syphilitic coryza.

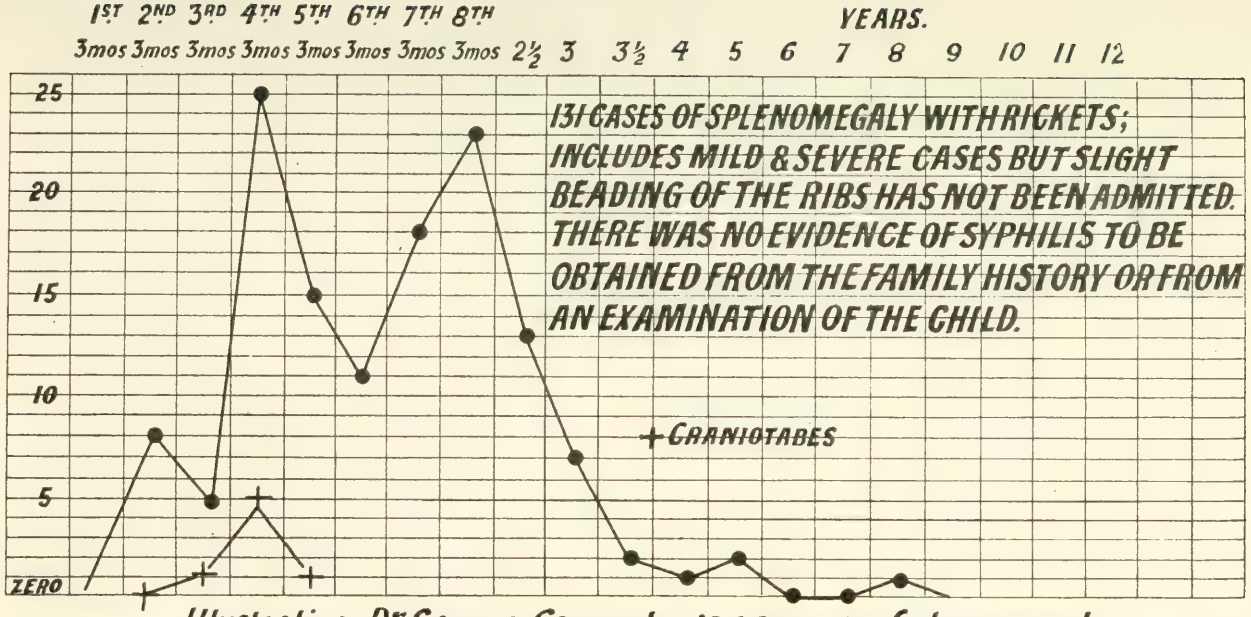
The points of interest about the condition to my mind are the facts that: (1), chronic "snuffles" undoubtedly occurs with great frequency in congenital syphilis,—that will be readily admitted by all; (2), that chronic "snuffles" and craniotabes have, in my experience, frequently been found associated; (3), that chronic "snuffles" and enlarged spleens are, as my figures demonstrate, not infrequent associates, with sometimes craniotabes in addition (*vide* Chart III); (4), that some cases of chronic "snuffles" are associated with adenoids, and that it is not always possible to say that the adenoids are the primary condition; that in a certain proportion of the cases which can be successfully examined digitally the naso-pharynx is quite free from adenoid growth; (5), that chronic "snuffles" is not always attended by a discharge from the anterior nares. The discharge is apt to trickle down the posterior pharyngeal wall and give rise to a reflex cough; (6), that chronic

"snuffles" when occurring alone may be of doubtful import, but when found associated with craniotabes, or splenic enlargements, or Parrot's nodes, or a combination of these or other conditions suspicious of syphilis, the balance of evidence is in favour of it being a syphilitic manifestation.

In Chart V the same plan of recording cases has been adopted, and this chart deals with 131 cases of splenomegaly associated with rickets, in which there was neither family history nor evidence on the person of syphilis. Of the rickety cases, it must be admitted at once that splenic enlargements are much more frequently met with in those who are undeniably rickety than in those who are only slightly so. In six cases only were the children *very* rickety. The existence of rickets, however, in cases of splenomegaly is more frequent than the chart gives credit for. As has been mentioned already, examples of rickets have arisen in the cases enumerated in Charts II, III, and IV. The total number with rickets would be 180, just a trifle over half the number of cases dealt with. Rickets is therefore slightly more in evidence than syphilis. At the utmost I can only arrive at a total of 147 cases of syphilis, whereas 180 children were rickety.

A study of this chart shows two strongly marked periods during this, the rickety age, viz. at and about the first and second years of life. For a reason which is not quite obvious, there appears to be a decline of rickets about the eighteenth month of life. Rickets is certainly most common at the first and second years of life. Cases begin to develop rapidly at the ninth month onwards, and to rapidly decline after the third year. Chart V also demonstrates how uncommon a feature craniotabes is during the rickety age, and how uncommon it is in the cases apparently free from syphilis. It will be noted that in a certain proportion of cases rickets occurs at from three to six months of age. In the absence of periosteal extension up the shafts of the bones it is difficult to separate the rickety joint from the

CHART 5.


1ST 2ND 3RD 4TH 5TH 6TH 7TH 8TH
3mos 3mos 3mos 3mos 3mos 3mos 3mos 3mos
YEARS.
2½ 3 3½ 4 5 6 7 8 9 10 11 12
25
20
15
10
5
ZERO
131 CASES OF SPLENOMEGALY WITH RICKETS;
INCLUDES MILD & SEVERE CASES BUT SLIGHT
BEADING OF THE RIBS HAS NOT BEEN ADMITTED.
THERE WAS NO EVIDENCE OF SYPHILIS TO BE
OBTAINED FROM THE FAMILY HISTORY OR FROM
AN EXAMINATION OF THE CHILD.
+ CRANIOTABES

Illustrating Dr George Carpenter's paper on Splenomegaly.

June, 1903.

Illustrating Dr George Ca

June. 1903

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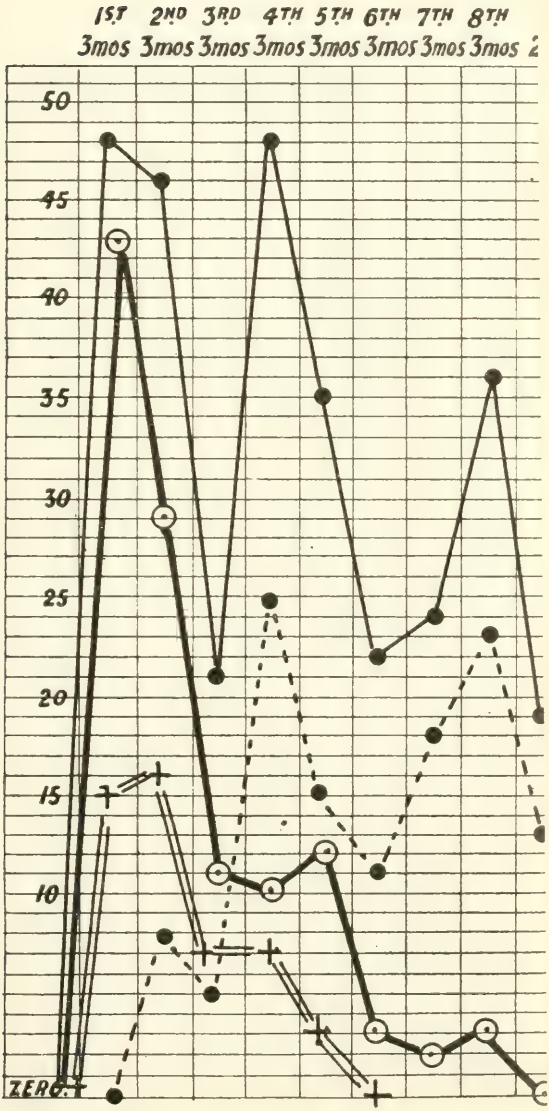

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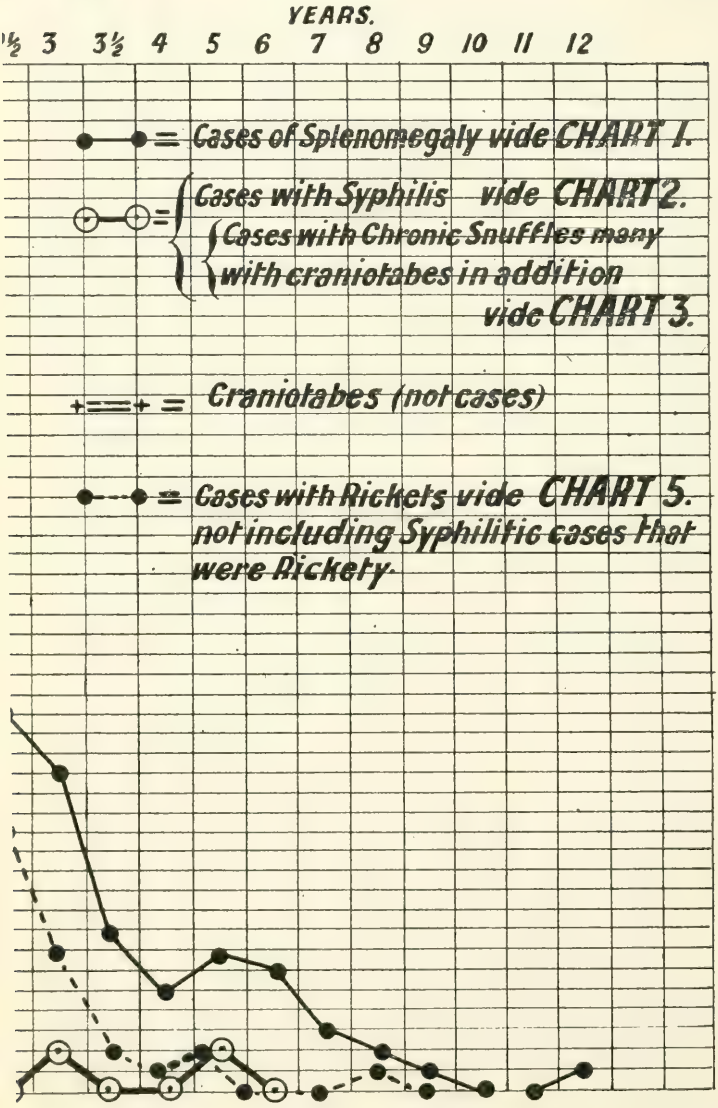
HART 6.


YEARS.
½ 3 3½ 4 5 6 7 8 9 10 11 12
= Cases of Splenomegaly vide CHART 1.
= Cases with Syphilis vide CHART 2.
Cases with Chronic Snuffles many with craniotabes in addition vide CHART 3.
= Craniotabes (not cases)
= Cases with Rickets vide CHART 5. not including Syphilitic cases that were Rickety.

syphilitic. Such enlargements occurring during the syphilitic age may possibly have been syphilitic, though they have been counted as rachitic. On the other hand, it will be seen that there were no cases of this condition during the first three months of life.

Chart VI is a composite chart which comprises all cases of splenomegaly, denoted by a thin line as in Chart I. It comprises cases of syphilis in Chart II united with cases of chronic "snuffles," many with craniotabes, shown in Chart III. These syphilitic cases are represented by a deep black line. Cases of rickets are represented by a broken line, and have been taken from Chart V. Chart VI also indicates the number of all these cases that had craniotabes, and these are represented by a double line. In this composite chart the thirty possible cases of syphilis depicted in Chart IV have not been included. It will be remembered, however, that seventeen of these thirty cases showed signs of rickets, the bulk of them being very definitely rickety. From this it would appear that all cases occurring within the first three months of life are probably syphilitic. During the next period of three months the great majority are certainly syphilitic, a few are possibly rickety, and six out of forty-six cases cannot be accounted for. During the next three months still the majority of cases are syphilitic, and there is but one case in which there is evidence neither of syphilis nor rickets. By the twelfth month syphilis has still further decreased and rickets still further increased. In three cases only is there evidence neither of syphilis nor rickets. By the fifteenth month in four cases only was there evidence neither of syphilis nor rickets, and rickets is a little more in evidence than syphilis. By the eighteenth month syphilis is not a strong feature, but rickets is pronounced. Four at least, however, cannot be accounted for. By the twenty-first month the majority are rickety; the remainder are syphilitic or are possibly so. By the second year the majority are rickety, and but three only cannot be accounted for. By two and a half years syphilis amounts

to a suspicion in one case only. All are rickety except six, which does not include the suspicious case, and three cannot be accounted for. By the third year there is trifling evidence of syphilis, and rickets is the more prominent of the two. Two cases could not be accounted for. By three and a half years two were rickety and three could not be accounted for. By the fourth year one was rickety, and one was doubtfully syphilitic, and one could not be accounted for. It further appears that during the rickety age, viz. from the ninth month to the third year of life, many more cases are associated with rickets than with syphilis. During the first four years of life the majority of cases can be included either under the headings syphilis or rickets.

There is a close connection in the chart between the rickety curve and the splenic curve, though not quite so intimate as that between the early syphilitic and the splenic; and the connection between the two is much closer between the latter half of the rickety period than during the first half. If the rickety cases in Charts III and IV were added the connection between the rickety and splenic curves would be more intimate still, and the syphilitic would sink somewhat by reason of the loss of Chart III.

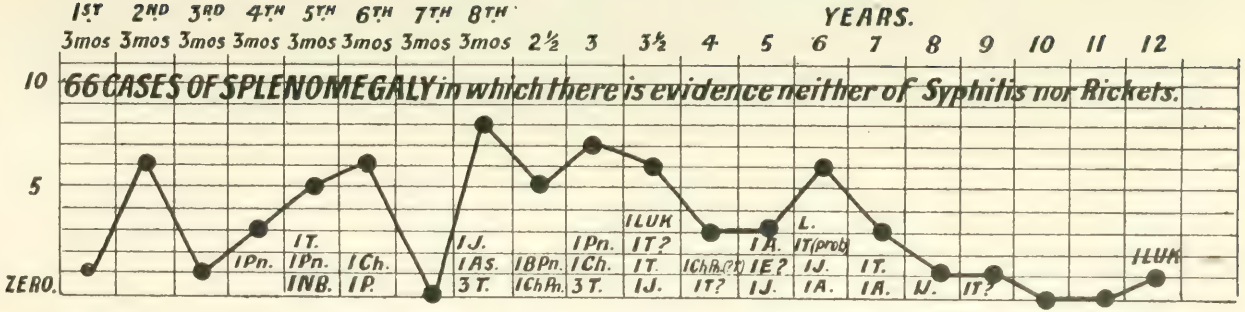
It will also be seen that syphilis, for the first fifteen months of life, is a not unimportant factor, though by the eighteenth month the zero line is closely approached, and syphilis does not materially affect the result after that period.

Splenic enlargement, however, is not a necessary associate of rickets; indeed, many cases of advanced rickets are free from enlargement of the spleen, and the great majority of rickety children have no splenic enlargement.

Chart VII deals with sixty-six cases of splenomegaly in which there was evidence neither of syphilis nor rickets. The diseases with which the enlargements were associated have also been recorded in many instances.

It is rare to find the spleen enlarged in general tuberculosis by reason of masses of tubercle within the organ.

CHART 7.



Pn=Pneumonia. NB=Twin Sister had Parrot's Nodes & was Rickety. Ch Pn = Chronic Pneumonia.
Ch=Congenital Heart. J = Jaundice. E = Enteric
T = Tubercle. As = Ascites. A = Ague.
P = Parrot's Nodes. B Pn = Broncho Pneumonia. L = Lymphadenoma.
LUK = Leukaemia.

Illustrating Dr George Carpenter's paper on Splenomegaly.
June. 1903

A tuberculous spleen like hardbake is rare, and tubercle in the spleen in quantity is uncommon. A grey or yellow tubercle here and there in the organ, with perhaps one or two in the capsule, is the common thing, and the enlargement of the spleen, when it is present in these cases has appeared to me to be mostly due to causes other than general tuberculosis.

The three cases of ague were imported from North Carolina.

I would call attention to the not infrequent association of catarrhal jaundice with splenic enlargement. This organ, like the liver, appears to become temporarily engorged in this disease.

It will be noticed that one of the cases in this group had Parrot's nodes, and another had a twin sister who was rickety and had Parrot's nodes.

There are, therefore, two important conditions of ill-health which are frequently associated with splenomegaly in infants and young children, viz.: (1), syphilis; (2), rickets; and there is (3), an undetermined cause or causes. At least two of the known conditions which accompany splenomegaly are admittedly in a high potential state during the first two or three years or more of life. Omitting the undoubted direct influence of syphilis in the production of splenomegaly—for it occurs in syphilitic fœtuses,—rickets is very prevalent at this period of infantile life-history.

Whether the alterations in the blood which induce rickets are capable of producing at the same time the splenic enlargements which undoubtedly arise in rickety subjects is not quite obvious, for the spleen is not usually increased in size in that disorder, and when enlarged the size of the spleen is in no sense, as it is in syphilis, a measure of the rickety cachexia. Nor is anæmia a necessary part of rickets. I have frequently observed *very* rickety children without any evidence of anæmia. Further, I have seen decidedly rickety and *anæmic* children develop splenic enlargements while under obser-

vation. I have also observed enlargement of the spleen where there had been rickets, but where that condition had disappeared. I have also seen rickety children with enlargement of the spleen become *more* rickety while the spleen diminished greatly in size. Splenic enlargement in rickety children is possibly, therefore, an epiphenomenon.

Rickets is not a disease the main feature of which is characterised by passive deprivation of the lime salts alone, but a strong feature of rickets is the active *overgrowth* of the cartilaginous growing ends, undue activity in the production of the scaffolding suited to a good building, with the deposition of jerry-built walls.

This undue activity at the growing ends and beneath the periosteum suggests an irritant, whatever may be the explanation for a temporary deficient ossification and undue softness of the bones,—of an irritant which is probably manufactured in the intestines, and is carried by the blood-stream to the skeleton.

Perhaps syphilis, which by reason of the state of ill-health which it produces is undoubtedly favourable to the occurrence of rickets, may be answerable for the production of some of the tardy splenic developments which occur in rickety infants. Thus one child under my care was very anæmic and very rickety, and had craniotabes. The craniotabes disappeared and gave place to Parrot's nodes* and an enlarged spleen, the child being rickety the while.

An explanation is still required for the undetermined cases which are free from syphilis, as far as it is possible to exclude that disease, and which are certainly not rickety.

The prevailing characteristics of the first two or three years of infant life are dietetic errors and gastro-intestinal disturbances. Defective hygienic environment among the poor is common to all ages of childhood, and is not peculiar to infancy; but unsuitable feeding and bowel troubles are the special features of this age,—of an age when the spleen is so prone to enlargement, of an age when rickets is so

* *Vide* another example, 'Reports of The Society for the Study of Disease in Children,' Postcript, p. 221, vol. iii.

common. It may well be that intestinal auto-intoxication, in the absence of other channels of septic absorption, or other causes for ill-health in the shape of previous severe illness, or by mineral or other poisoning, may be held responsible for splenomegaly. That continued bowel troubles in infants are provocative of rickets is undoubted; so, too, are they of splenomegaly, but it will not do at present to push the analogy too far, for the evidence says nothing as to whether the poisons which produce rickets, splenomegaly, and anæmia are identical or differ among themselves. These several conditions are associated by reason of the time of life at which they happen to be prevalent, but I take it that they might be due to a diversity of causes, all of which, however, may emanate from the bowels. As I previously remarked, it appears to me that splenic enlargement in rickets is an epiphenomenon.

But, in this group also there are difficulties in the way of excluding syphilis. Thus I have seen a pallid, "snuffling" infant, with marked craniotabes which was cured by mercury. Some months later, while still under mercurial treatment and still very anæmic, but without leucocytosis, it developed a large spleen, and it was free from obvious rickets. It subsequently became fat and rosy-looking when arsenic was added to its medicine. The spleen, nevertheless, remained unchanged. A year later the spleen was normal. Arsenic, cod-liver oil, and iron had been taken in the meanwhile. Here are other examples:—Twin infant sisters had greatly enlarged spleens. One of these was non-rickety, and syphilis could not be detected. Anæmia became extreme without leucocytosis, and she died (*vide* Case No. 17, page 316). The other twin was a little rickety, lost its anæmia, became more rickety the while, and developed Parrot's nodes, the spleen still remaining full large. In another set of twin infant sisters, in whom there was evidence neither of rickets nor syphilis, in one of them the spleen was enormously enlarged, while that of the other was quite natural. The feeding and environment of both were identical. I have also seen splenic

enlargements under similar hygienic conditions in young children of the same family.

The blood.—Children with splenomegaly are often not obviously anæmic. Their colour ranges from a doubtful pallor through varying gradations to a milk-and-water hue with a bluish tinge about the lips, or to a lemon-yellow appearance. Petechiæ and bruises are not uncommon in the very severe cases, and these often herald a fatal termination, but not invariably. Some of these severe cases are the subject of lichen urticatus, a not uncommon complication of splenomegaly, and I have in them sometimes noticed the occurrence of hæmorrhages into the urticarial lesions. Œdema of the extremities and occasional albuminuria were noted in some of the fatal cases.

The cases can be conveniently classified under several headings.

(1) Cases of deficient hæmoglobin. The hæmoglobin was deficient, and ranged from 82 per cent. to 47 per cent. The red corpuscles were increased in quantity from 100 per cent. to 140 per cent. The colour index ranged from ·4 to ·7. The ratio of white corpuscles to red varied between 1 to 1300 and 1 to 480. The films were normal.

(2) Cases where the hæmoglobin was deficient and the red corpuscles were reduced in number. Sometimes the red corpuscles varied in size, and poikilocytes were seen together with granular degeneration of the red corpuscles and polychromatophilia. Normoblasts and megaloblasts were occasionally seen. The white corpuscles were not increased in number, but an occasional myelocyte was observed.

(3) Cases similar to No. 2, and also some with more advanced changes in the red corpuscles, in which there was polymorphous leucocytosis of varying degree, and sometimes it was very extensive. It was not common to find much more than about 10 per cent. of myelocytes.

(4) Two cases in which the hæmoglobin was very low, viz. 13 per cent. and 18 per cent., and in which the red corpuscles were greatly reduced in number, viz. to 19 per cent. and to 55 per cent. Microscopically many nucleated red blood-corpuscles were seen together with megaloblasts. Microcytes,

macrocytes, and poikilocytes were found, and polychromatophilia. There was polymorphous leucocytosis in the first case, 1 white to 66 red corpuscles, and a few myelocytes were seen in the other.

(5) I have two examples of leukæmia in young children; one was *lymphatic*, and the other *spleno-myelogenous*. In both forms the hæmoglobin was greatly reduced, and also the red corpuscles. In the former the red corpuscles were unchanged, in the latter the films showed poikilocytes, macrocytes, and microcytes, and numerous normoblasts and megaloblasts and karyokinesis. In the former the lymphocytes were 99·18 per cent., mainly the small lymphocytes. The polymorphonuclear leucocytes were greatly diminished. A few eosinophiles and myelocytes were seen. Dr. McCaw, of Belfast,* has recently reported a case to this Society in a non-rickety and non-syphilitic infant eighteen months old. The lymphocytes reached 99·2 per cent., and a few nucleated red corpuscles were seen. In the spleno-myelogenous infant the myelocytes amounted to 21·4 per cent. of the leucocyte count, the large and small lymphocytes to 31·5 per cent., and the polymorphonuclear to 46·5 per cent.

The fact that the spleen is very ready to enlarge during the first two years or more of life is sufficiently obvious, but the part played by the enlarged organ in these cases of splenomegaly does not seem very clear.

Anæmia may be extreme and yet there be no splenic enlargement; and, on the other hand, quite an appreciable enlargement of the spleen is frequently associated with a very mild form of anæmia. Nor does an inquiry directed to the supposed physiological functions of the spleen in correlation with the known pathological conditions of the blood assist in the inquiry. The post-mortem findings are equally unsatisfactory, as there is nothing characteristic about the enlarged organ whether it be in the case of a syphilitic or not. Interstitial changes and gummata are great rarities, and then are usually found after two years of age. Aside from the fact that an enlarged spleen appearing during the first six months of life, even when taken alone, is very suggestive of syphilis, splenic en-

* 'Reports of The Society for the Study of Disease in Children,' vol. iii, p. 251 – 255.

largements, as such, in infants and young children appear to me to be of no great clinical moment, and though they may point to syphilis in some cases, there is no justification in ascribing them to that disorder in the absence of corroborative signs; their importance in any event lies in the accompanying condition of the blood, and without an examination of this it is impossible to form an opinion as to the future prospects of the infant.

The liver is enlarged in the great majority of the cases of splenomegaly. Sometimes this is quite trifling, but it often keeps pace with the size of the spleen,—that is, it is one or two or three or more fingers below the costal margin in the nipple line. It is sometimes much more below the costal margin than the spleen is in front of it. Induration of the organ in the first few months of life suggests syphilitic infiltration, and when there is jaundice and bile-free stools from birth there is congenital malformation or destruction of the bile-ducts, which may be syphilitic. Ascites may be a prominent feature. In later childhood induration of the liver with splenomegaly suggests cirrhosis of the liver. Enlargement of the liver combined with splenomegaly from catarrhal jaundice; chronic heart disease, congenital and acquired; lymphadenoma; leukæmia; lardaceous disease; and ague are not uncommon.

The lymphatic glands.—A trifling enlargement of some of the accessible lymphatic glands is not uncommon.

Prognosis.—This depends upon the condition of the blood. A great reduction in the hæmoglobin and in the number of the red cells, especially if there be great changes in them in size and form, is not hopeful. Leucocytosis, apart from its association from a mild form of anæmia, especially if it be in considerable amount, renders the outlook less promising. At all times such children are liable to succumb to broncho-pneumonia. In pernicious anæmia the preponderance of megaloblasts over normoblasts is of bad omen. The changes in the blood characteristic of leukæmia appear almost invariably to point to a fatal termination.

Treatment should be preventive—the provision of proper food and a hygienic environment. Rickets and syphilis require appropriate treatment. The anæmia should be dealt with by fresh air and good food and the correction of bowel complaints. Of drugs, iron and arsenic were used, as also the inhalation of oxygen over prolonged periods, and they certainly appeared to do good in many cases. Hæmatogen has also proved of some benefit, but I have not seen any advantage derived from bone-marrow.

Appended are a few illustrative cases. I have to express my indebtedness to Dr. David Nabarro for his valuable assistance in working out the blood changes in the bulk of these cases.

1. Dorothy S—, aged 7 months. Ribs beaded, ankles ridged. Spleen one and a half fingers, and liver two and a half fingers below costal margin. "Snuffles" ever since birth more or less; no adenoids.

Hæmoglobin 60 per cent.; red corpuscles 7,000,000 per c.mm., 140 per cent.; colour index ·43. White corpuscles 9000 per c.mm., ratio 1 to 780. Films normal. No nucleated red corpuscles. Leucocytes normal. The infant died some three weeks later, its death being preceded by some gastric disturbance.

2. Eva S—, aged 3 years. Rickety. Spleen two fingers below costal margin, and liver large but not hard. No history of syphilis. She is somewhat bronzed about her abdomen. Jaundice at the age of two years. Has recently been in the hospital with pneumonia. Not anæmic-looking.

Hæmoglobin 82 per cent.; red corpuscles 6,000,000 per c.mm., 132 per cent.; colour index ·62. White corpuscles 12,400 per c.mm., ratio 1 to 532.

3. Dorothy S—, aged 1 year 11 months. Decidedly rickety; no evidence of syphilis; bottle-fed. Spleen one finger, and liver two fingers below the costal margin. A little pale, but this is not a marked feature.

Hæmoglobin 62 per cent.; red corpuscles 6,500,000, per c.mm., 130 per cent.; colour index ·5. White corpuscles 5000 per c.mm., ratio 1 to 1300.

4. Phyllis C– , aged 2 years and 1 month. Not rickety, but pot-bellied. Not syphilitic. Accessible lymphatic glands shotty. Spleen one finger and liver two fingers below the costal margin. Accessible lymphatic glands shotty. Anæmic; three small bruises front of left shin.

Hæmoglobin 47 per cent. to 48 per cent.; red corpuscles 6,000,000 per c.mm., 120 per cent.; colour index ·4. White corpuscles 10,200 per c.mm., ratio 1 to 588. Films show nothing abnormal. Six weeks later it was a better colour, but it was sick in the morning and its bowels were relaxed, and it was thought to be losing flesh. The spleen was smaller.

5. William W—, aged 6 months. Beaded ribs obvious; wrists *plus*, and ankles *plus*. Chronic "snuffles," a discharge from the nose, and "child crowing." Spleen one finger and liver half a finger below the costal margin.

Hæmoglobin 75 per cent.; red corpuscles 5,500,000 per c.mm., 110 per cent.; colour index ·7. White corpuscles 11,300 per c.mm., ratio 1 to 490. The films look normal. The white corpuscles are not increased; excess of lymphocytes present.

6. Lilian S—, aged 10 months. Beaded ribs; wrists *plus*. Craniotabes right parietal bone; bottle-fed. "Snuffles" now, and when she was born a little. Spleen two fingers and liver just below the costal margin. Glands in the groin shotty. A trifle pale. "Sick at everything."

Hæmoglobin 56 per cent.; red corpuscles 5,400,000 per c.mm., 108 per cent.; colour index ·52. White corpuscles 7200 per c.mm., ratio 1 to 750.

7. Ivy D—, aged 1 year and 4 months. A little rickety, perhaps. Three still-births; bottle-fed. Thin and bad-tempered. Spleen one finger and liver half finger below the costal margin. Pale.

Hæmoglobin 60 per cent.; red corpuscles 5,217,000 per c.mm., 104 per cent.; colour index ·6. White corpuscles 10,800 per c.mm., ratio 1 to 480. Films normal.

8. Louisa F—, aged 1 year and 3 months. Not rickety, and no evidence on the person of syphilis, but four children were born dead, all before this one. "Thrush," but not to the calves. Spleen two fingers and liver two fingers below the costal margin. A good deal of post-nasal catarrh, but nothing to be felt digitally. Pale, but not specially so. The erythrocytes are normal in size

and shape, but display a well-marked central pallor. There is no leucocytosis. Differential count. Small lymphocytes 22·6, large 8·0; total 30·6. Polymorphonuclear 69·0, eosinophiles 0·4; total 100. The child made two or three attendances and improved somewhat. It then developed pneumonia, and no further attendance was made. It probably died.

9. Lilian C—, aged 2 years and 7 months. Rickety. Well-marked parietal osteophytes, but no evidence of syphilis otherwise; a very suspicious history, however. Spleen one finger and liver two and a half fingers below the costal margin. Accessible lymphatic glands normal. Feeding bad. Very pale.

Hæmoglobin 28 per cent.; red corpuscles 3,200,000 per c.mm., 64 per cent.; colour index ·44. White corpuscles 4000, 40 per cent., ratio 1 to 310. Films. Reds vacuolated, very variable in size, especially macrocytes present. No polychromatophilia; no great poikilocytosis; no nucleated reds. White corpuscles; large hyaline corpuscles, unusually large. Oxyphilic, fine (polymorphonuclear) 21·7 per cent., equals 872 per c.mm.; coarse (eosinophilous) 2·4 per cent., equals 96 per c.mm. Mononuclear (small lymphocytes) 51·9 per cent., equals 2076 per c.mm.; (large hyaline) 18·8 per cent., equals 772 per c.mm. Basophilic (fine), 0·5 per cent., equals 20; coarse (Mastzellen) 1·5 per cent., equals 60 per c.mm. Myelocytes, all finely oxyphilic, 3·5 per cent., equals 150 per c.mm.

10. Ethel V. K—, aged 1 year and 4 months. Rickets is not a marked feature. Twin to Ivy who died (*vide* Ivy K—). Lips a little pallid. Spleen three fingers and liver two fingers below the costal margin. Accessible lymphatic glands a little enlarged. Fundus oculi normal but pale.

Hæmoglobin 42 per cent. Red corpuscles 3,400,000 per c.mm., 68 per cent.; colour index ·6. White corpuscles 6500 per c.mm., ratio 1 to 523. Films nothing abnormal, no nucleated red corpuscles and no myelocytes. Eighteen days later, under arsenic she was better and the spleen was not so tender. A tendency to Parrot's nodes was now noticed.

Five months later the tendency to Parrot's nodes had grown into distinct parietal bosses with an intervening groove. She was not anæmic, but very decidedly rickety; the spleen was a finger's breadth less in size.

Hæmoglobin 84 per cent. to 86 per cent. Red corpuscles

5,000,000 per c.mm., 100 per cent.; colour index ·85. White corpuscles 10,000 per cmm., ratio 1 to 500. Differential count (1000): small lymphocytes 49·5 per cent.; large lymphocytes 15·5 per cent.; polymorphonuclear 34·7 per cent.; eosinophiles 0·7 per cent.; basophiles 0·1 per cent.; no myelocytes. It was seen three months later, and was then "still further improved."

11. Henry C—, aged 10 months. Obviously rickety, craniotabes right parietal and right side of occipital bones. No evidence of syphilis. Bottle-fed. Spleen one finger and liver half a finger below the costal margin.

Hæmoglobin 75 per cent. Red corpuscles 3,800,000 per c.mm., 76 per cent.; colour index 1. White corpuscles 11,300 per c.mm., ratio 1 to 336. Greatly improved under cod-liver oil and hæmatogen.

12. William C—, aged 1 year and 2 months. The chest was pinched-in a trifle below the nipple level, otherwise no evidence of rickets. No evidence of syphilis. Twin sister was healthy, and had no splenic enlargement. Spleen four fingers below the costal margin, reaching close to umbilicus and liver, two and a half fingers below the costal margin. Accessible lymphatic glands a trifle enlarged. Fundus oculi normal. Pale, complexion sallow.

Hæmoglobin 50 per cent.; red corpuscles 4,100,000 per c.mm., 82 per cent.; colour index ·6. White corpuscles 8200 per c.mm., ratio 1 to 500. Films, no abnormal corpuscles seen; no myelocytes. Proportions of leucocytes apparently unaltered (normal).

13. Mary B—, aged 2 years and 5 months. Not rickety. Pot-bellied and chest pinched in below the level of the nipples, and may therefore have been a trifle rickety. Spleen one finger and liver three fingers below the costal margin. Accessible lymphatic glands a trifle enlarged. Pale.

Hæmoglobin 58 per cent. to 59 per cent.; red corpuscles 4,800,000 per c.mm., 96 per cent.; colour index ·6. White corpuscles 108,000 per c.mm., ratio 1 to 444. Films normal.

14. George W—, aged 1 year. Rickety a little. No history or evidence of syphilis. Breast-fed. Analysis of mother's milk by Dr. Pakes: total solids 5·83, fat 2·39, sugar 2·40, proteid 1·12. Spleen one finger below the costal margin; feels rather hard.

Accessible lymphatic glands shotty. Very pale, a lemon-yellow colour. Two purpuric spots in front of right ankle.

Hæmoglobin 13 per cent. to 14 per cent.; red corpuscles 950,000 per c.mm., 19 per cent.; colour index ·7. White corpuscles 14,400 per c.mm., ratio 1 to 66. Films, red corpuscles all sizes and shapes. Some megaloblasts with polychromatophilia. White corpuscles all kinds, ? myelocytes. There were no pathogenic germs in the milk. He was ordered arsenic. The following day he developed diarrhœa and sickness, and the next day he died.

15. Rose S—, aged 5 months. Ribs distinctly beaded, wrists *plus* and ankles *plus*. Extreme craniotabes at the posterior part of the right parietal bone. "Snuffles" when born, which "lasted up to three weeks ago." Has a frequent cough due to discharge at naso-pharynx. Spleen one finger below the costal margin and liver one finger also. A trifle pale.

Hæmoglobin 60 per cent.; red corpuscles 2,950,000 per c.mm., 59 per cent.; colour index 1. White corpuscles 36,500 per c.mm., ratio 1 to 80. Films look normal. Red corpuscles in rouleaux. White corpuscles considerably *plus*. Ordered hæmatogen. He attended for a short time and was better.

16. William T—, aged 1 year. Very rickety. Craniotabes right parietal bone. "Snuffles" ever since birth. Premature child at seven and a half months. Spleen normal. Very anæmic. Treated by mercury. In spite of mercury spleen found to be enlarged six weeks later, and Parrot's nodes were commencing. He was then very pale. Spleen two fingers and liver three fingers below the costal margin.

Hæmoglobin 50 per cent.; red corpuscles 4,100,000 per c.mm., 82 per cent.; colour index ·6. White corpuscles 25,000 per c.mm., ratio 1 to 164. Films, blood-platelets apparently increased.

Four months later he was admitted into hospital with bronchopneumonia, and the blood was again examined.

Hæmoglobin 50 per cent.; red corpuscles 4,500,000 per c.mm., 95 per cent.; colour index ·52. White corpuscles 11,800 per c.mm., ratio 1 to 380. Films, red corpuscles vary somewhat in size; no nucleated red corpuscles. White corpuscles normal, ? few myelocytes. The spleen was still enlarged.

17. Ivy K—, aged 1 year and 2 months, not rickety. Twin sister of Ethel V. K—, who was rickety and had cranial osteophytes. Rather depressed bridge to nose. Wasted. Spleen four fingers and liver three fingers below the costal margin. Accessible lymphatic glands palpable. Very pale, milk-and-water appearance.

Hæmoglobin 16 per cent. to 18 per cent.; red corpuscles 2,750,000 per c.mm., 55 per cent.; colour index ·33. White corpuscles 16,000 per c.mm., ratio 1 to 172. Films, a few myelocytes. Many nucleated red corpuscles, especially megaloblasts, some with mitosis. Polychromatophilia. Microcytes, macrocytes, and poikilocytes. Was treated with arsenic, but did not improve, and died twenty-six days later. Crepitations were heard at both bases before death.

Post-mortem.—Stomach extremely contracted, capacity half an ounce. Lungs, broncho-pneumonia. Spleen large, weighed three and three-quarter ounces, normal in appearance. Marrow of femur red, soft, and jelly-like.

18. Sydney B—, aged 1 year and 4 months. He is rickety, but this is not a marked feature. Abdomen rather pot-bellied. Spleen two fingers and liver 1 finger below the costal margin. Accessible lymphatic glands a trifle enlarged. Pale decidedly. The gums round the lower central incisor teeth are reddened. Petechiæ all over.

Hæmoglobin 50 per cent.; red corpuscles 3,144,500 per c.mm., 63 per cent.; colour index ·8. White corpuscles 17,950 per c.mm., ratio 1 to 175. Films, the red corpuscles vary much in size; some poikilocytes; granular degeneration of red corpuscles. Polychromatophilia. Nucleated red corpuscles present. An occasional myelocyte seen. Ordered hæmatogen, and improved under it.

19. Ethel M. R—, aged 1 year and 2 months. Very rickety. Parrot's nodes. Chronic "snuffles" (adenoids); the post-nasal examination gave him a cough like whooping-cough, and after the cough he was puffy about the eyes. Trace of albumen in the urine. Accessible lymphatic glands shotty. Optic discs probably too white. Spleen three fingers and liver two fingers below the costal margin. Pale.

Hæmoglobin 40 per cent.; red corpuscles 3,400,000 per c.mm., 68 per cent.; colour index ·6. White corpuscles 17,000 per

c.mm., ratio 1 to 200. Differential count: small lymphocytes 38·5; large lymphocytes 16·3; polymorphonuclear 42·4; eosinophiles 2·1; basophiles 1·0. Twenty nucleated red corpuscles were seen while counting 370 white corpuscles; there were a few megaloblasts. He developed epistaxis, and died nearly a month after the first attendance. There was no change in the spleen. Arsenic and cod-liver oil were given.

20. Leonard P—,* aged 3 years and 6 months. Neither rickety nor syphilitic. Became pale five weeks previously, suddenly. Spleen from two to three fingers and liver two fingers below the costal margin. No obvious enlargement of lymphatic glands anywhere. Extreme pallor, mucous membranes pale. A few bruises on the legs. A trace of albumen.

Hæmoglobin 15 per cent.; red corpuscles 1,568,000 per c.mm., 31 per cent.; colour index ·5. White corpuscles 130,000 per c.mm., ratio 1 to 12. Fresh films show red corpuscles in rouleaux; no microcytes or poikilocytes. Differential count of 4000 leucocytes: small lymphocytes (mainly) and large lymphocytes together 3967, equals 99·18 per cent.; polymorphonuclear 19, equals ·48 per cent.; eosinophiles 5, equals ·12 per cent.; myelocytes (one eosinophile myelocyte) 9, equals ·22 per cent. One nucleated red corpuscle was seen while counting 4000 leucocytes.

Post-mortem.—Lymphoid infiltration of the liver in the portal canals. Spleen 4 oz., normal in appearance. Kidneys enlarged and leucocytal infiltration. Tuberculous intestinal ulceration and caseating mesenteric glands.

21. Henry P—, aged 1 year. Neither syphilitic nor rickety. Bottle-fed. Wasted and anæmic; petechiæ. Spleen four fingers and liver two fingers below the costal margin.

Red corpuscles 2,900,000 per c.mm. White corpuscles 48,000, ratio 1 to 60. Differential count: small lymphocytes 23·3 per cent.; large lymphocytes 8·2 per cent.; myelocytes 21·2 per cent.; polymorphonuclear 46·7 per cent.; eosinophiles ·6 per cent. Films, poikilocytes, microcytes, and macrocytes. Normoblasts and megaloblasts. Karyokinesis.

(*Read at the Provincial Meeting, Brighton, June the 20th,* 1903.)

* 'Reports of The Society for the Study of Disease in Children,' vol. iii, pp. 47–50.

66. PRIMARY PNEUMOCOCCIC MENINGITIS.

By EDMUND CAUTLEY, M.D.

Pneumococcic meningitis is usually secondary to affections of the lungs or pleura. Such cases are generally coincident with the primary disease, very severe and rapid in their course, and occur only a few days before death. The cerebral symptoms may be quite insignificant, or may be entirely masked by those of the primary disease. The presence of the meningitis, even though very extensive, may be only ascertained at the autopsy.

More rarely the disease is primary, unaccompanied by any disease in the chest. Such cases are generally severe and rapidly fatal, and we rarely meet with them in a benign form. Possibly, however, the benign form is not as rare as is supposed. It may remain undiagnosed because of the slightness of the meningitic symptoms. Or it may be diagnosed as a sporadic case of cerebro-spinal meningitis, from lack of the confirmatory evidence which might be obtained by lumbar puncture and bacteriological examination of the fluid obtained.

I have brought this case forward to illustrate these points, which may be summed up shortly, viz., the slight character of the meningitic symptoms, the course of the fever and its termination by crisis, and the uncertainty of the diagnosis, as to the presence of meningitis and its variety, until the results of lumbar puncture were obtained.

William J—, aged 9 months, was admitted into the Metropolitan Hospital under my care on March the 19th, 1903. The family history and past history were unimportant. The child took ill on March the 17th with fever, vomiting, and retraction of the head. Next day he had diarrhœa, with offensive stools. On admission he was found to be much undersized, weighing 10 lbs. 12 oz., and had no teeth. The pupils were equal; no squint. The fontanelle was open, tense and pulsating. There was some

retraction of the head, with rigidity of the neck muscles. A few scattered râles were heard in the lungs. The abdomen was rather distended, no diarrhœa, no tâche cérébrale. The knee-jerks were present and Kernig's sign was absent.

March the 20th.—Constant crying. Takes food fairly, occasional vomiting, costive. Tense bulging fontanelle. Retracted head. No squint; pupils small, right a little the larger. Full abdomen. Brisk knee-jerks, normal plantar reflex. Pulse-rate 144 to the minute, regular. Respirations 40 to 60 to the minute. Temperature 103° F. to 105° F.

Lumbar puncture was performed in the afternoon. One ounce of clear fluid was withdrawn, drop by drop, running easily. Microscopical examination showed capsulated diplococci. Cultures on agar agar yielded a growth of diplococci, not decolourised by Gram's process. (Report by Mr. Gordon Watson, pathologist to the hospital.)

21st and 22nd.—No appreciable change. Higher fever.

23rd.—Considerable fall in temperature.

24th.—Temperature down in the morning, high at night. Loss of 8 ounces in weight since admission into the hospital. Fontanelle less tense. Child brighter, occasionally vomits.

25th.—Temperature subnormal in the morning and high at night.

April the 3rd.—Temperature fell during the night of the 25th and has remained down since. From March the 24th to the 31st the child increased 8 ounces in weight, but has not gained since. Takes food well. Convalescent.

In the afternoon the head was again a little retracted but the fontanelle remained flat. Next day an occasional squint was noted and it was thought that the child was going to have a relapse. The following day there was more head retraction; discharges from the eyes and nose, and moderate fever were also present. On April the 6th the nasal discharge was more profuse, the temperature higher, and the fontanelle bulging. On the 7th the nasal discharge was thick and

yellow. The child vomited three times and had lost 9 ounces in weight during four days. Lumbar puncture on the 8th yielded one ounce of clear, sterile fluid. The child was worse, taking food badly, and vomiting. On the 10th the nasal discharge was more profuse and blood-stained. Membrane was found on the uvula and the left tonsil. He was transferred to a fever hospital and there died. No autopsy was obtainable. Had it not been for the unfortunate development of diphtheria, the child would have been discharged as cured.

Remarks.—The course of the fever, its duration and mode of termination are very similar to those of a simple croupous pneumonia. It ended on the tenth day and the temperature remained down, just as in pneumonia. The appearance of the child was never in the least suggestive of pneumonia. The chest was carefully examined and no evidence of consolidation could be found. Nothing abnormal was ever found in the chest, except a few râles. There was neither cough, nor flushing of the cheeks, nor hot dry skin, nor delirium. In fact, the child never seemed dangerously ill. The pulse-rate and respirations were increased in proportion to the height of the fever. The breathing was very frequent, but that is common in high fever in infants. The pulse was never irregular.

The diagnosis of meningitis was based upon the fever, vomiting, and retraction of the head. It was confirmed by lumbar puncture. There was no optic neuritis.

Lumbar puncture was also beneficial in reducing the intra-cranial tension, and possibly by withdrawing diplococci and toxins. Its value is not sufficiently realised in the diagnosis, treatment and prognosis of cerebral cases in children. If adopted for treatment it should be begun early and repeated often. It is probably the only therapeutic method of value at our disposal in this class of case, beyond careful attention to feeding and nursing. Possibly anti-pneumococcic serum may ultimately prove of value.

(*Read at the Provincial Meeting, Brighton, June the 20th*, 1903.)

Discussion.

The Chairman (**Dr. Wayland Chaffey**) (Brighton) said Dr. Cautley's case reminded him of one by the same author recorded in the last volume of the Society's 'Reports' In that paper there was a very exhaustive account of meningitis in its various forms, especially in dealing with the bacteriology. It showed an advance in our knowledge of the subject.

Dr. Theodore Fisher (Bristol) said he remembered a case in which there was retraction of the head, and other signs, and lumbar puncture was performed during life, but in which no evidence of meningitis was found post mortem. There was, however, thrombosis of some of the cerebral veins. The case was recorded in the 'British Medical Journal' some time ago. There might be head retraction associated with peumonic infection, due to three things: (1), the cerebral symptoms might be due simply to toxæmia; (2), to the entrance of micro-organisms into the blood and cerebro-spinal fluid; and (3), to meningitis itself. They might be regarded as three stages of the same condition.

Dr. G. A. Sutherland asked whether there was any ear trouble associated with the illness.

Dr. Edmund Cautley, in reply, said there was no ear trouble, and the child never seemed very ill, in spite of the fever and head retraction. Except for the results of lumbar puncture, one would have regarded it as a mild case of that type of cerebro-spinal meningitis of which one saw cases recover. He was practically certain there was no consolidation of the lung at any spot, and there were very few râles in the chest.

67. THE USE OF OPIUM IN THE CARDITIS OF CHILDREN.

By ALEXANDER MORISON, M.D.

Following the late Dr. Octavius Sturges in the use of the general term carditis as expressing the frequently observed phenomenon of the implication in the inflammatory process of more than one, and it may be of all the structures of the cardiac wall, I shall briefly sketch the irritative

conditions which this term denotes, before referring to their treatment by opium.

Normal cardiac action, especially in the higher animals, being due to the harmonious co-operation of the hæmic, muscular and neural factors in such action, it follows that morbid action is a result of the disturbance of this harmony by agencies affecting one or all of these factors.

As the blood bathes all the structures of the heart its invasion by disease-producing agencies naturally affects all the structures which are dependent upon it for their nutrition. Bacteriology has taught us that the blood circulating in the chambers and in the vascular system of the heart may carry such morbific agencies, and thus be the cause of those secondary effects which we find expressed in the inflammation of the various constituents of the organ.

The aim of the physician should, doubtless, be to remove causes, and having done so to expect effects to disappear. But, great as have been the triumphs of therapeutic bacteriology in some spheres, patients still die from the effects even of toxic agents of the nature of which we know somewhat, and also from the effects of those of which we know little or nothing, except that certain effects produced by them prove fatal to patients. Were we under these circumstances to wait for the discovery of causes before endeavouring to cope with known effects, we should be acting neither rationally nor humanely. Effects as well as causes, however, point the way to rational treatment, for effects are themselves the secondary causes of still further disaster, and life is usually lost, not when the first outworks are stormed by an unknown or invisible enemy, but when rampart after rampart has been taken and the central keep itself assailed by enemies whom it is easy to see if we look for them. Not, that is, by the first onslaught of a primary cause, but by the pernicious influence of effects which may be laid bare to the eye by macroscopic and microscopic examination.

These effects in carditis may be seen in all the structures of the heart, and consist, in the main, of all degrees of

inflammation recent and of longer standing of the vessels, nerves, interstitial textures, muscle-cells, and serous surfaces of the organ. Of these, the serous surfaces and interstitial textures seem to be those chiefly affected in the majority of cases, the nerves and muscle-cells suffering as a rule from the inflammation of these until all the cardiac textures may be involved in a general conflagration.

Indeed, just as the study of reflection in a rain-drop may explain the glory of the rainbow, so also inflamed blood-vessels, such as may be found in the epicardium of a heart affected by pericarditis, may epitomise the processes taking place in the organ generally. Thus, as in a specimen I show, the thickened fibrous coat of the artery, the abundantly proliferating intima, and the apparently increased muscular tissue of the vessel with degenerated nuclei may be regarded as corresponding to the inflammatory, hypertrophic and degenerative processes which are observed to affect, in many cases, all the textures of the organ from the endocardium to the inflamed or it may be adherent pericardium. To wait for the discovery of a rational means of coping with first causes would, under these circumstances, be irrational. As Dr. Cheadle has with practical sagacity remarked, in one of his lectures, "while the grass grows the steed may starve;" while the physician holds his hand waiting for scientific enlightenment the patient may die, if we do not promptly grapple with death-dealing effects or secondary causes, if we do not, that is, gain from signs and symptoms the cue to successful interference.

In the treatment of carditis, to the minds of most physicians of to-day, two main facts have become apparent, viz.: (1), the uselessness of adding fuel to the fire by irritation or counter-irritation; and (2), the desirability of allaying by all means at our command local and general irritation and unrest. In acting on such lines the important *rôle* played by the central and peripheral nervous system scarcely requires to be emphasised. And yet, the condition of the cardiac nervous system is rarely investigated by the

pathologist. When one considers the rich endowment of the heart by nerves, and remembers the anatomical situation of the main streams of these in the intermuscular connective tissue, in those very parts in which we meet with the most marked evidences of inflammation, we cannot avoid the conclusion that agents which relieve irritative states of these governing channels of cardiac action are calculated to be most useful in dealing with such states.

In the micro-photographs which I have placed on the table the course and distribution of the cardiac nerves may be observed surrounded, if not actually affected, by actively proliferating connective tissue in the specimens prepared from fatal cases of carditis. The staining of the finer nerves in specimens taken from the dead body is not an easy matter. I have therefore placed among the pathological specimens a Golgi preparation which I made from the heart of a cat which was anæsthetised and drowned. I was for a time inclined to regard the rich innervation here shown with some scepticism, regarding it as possibly the chrome silver staining of elastic tissue, but having stained the intermuscular bundles by Nissl's methylene blue method, and having traced the nerve to its origin under the epicardium, as is well shown in one of the specimens exhibited, I am confirmed, in my opinion, that the Golgi preparation shown exhibits nervous structures. If this be granted, we can well conceive how important a part in causing cardiac unrest may be played by the intrinsic nervous system of the heart when stimulated by an interstitial myocarditis.

Indeed, the efficacy of those measures found to be most beneficial in the treatment of carditis seems to point the same lesson, as does the inutility of agents found of much use in cases of non-inflammatory cardiac failure. Thus, we now know that it is altogether questionable whether the salicylates are an antidote to the rheumatic poison, but we have no doubt that no other agent so rapidly relieves the pain associated with that process and thus indirectly calms the patient as a whole, and with this general relief the

irritability of his heart also. Locally applied ice in those cases in which it can be borne, in the virtues of which Dr. W. B. Lees strongly believes, can also affect the heart only through the nervous system, whether such effect be regarded as directly sedative to the nervous structure or astringent to the inflamed blood-vessels. The relief of pain by the local abstraction of blood is likewise an oft-observed phenomenon, and when relief is thus obtained by the application of a few leeches to the precordia it can only act by relieving the irritation of nervous structures, for the amount of blood withdrawn can have little effect on the general circulation. But, useful as leeching and the application of ice may prove in the case of adults and in some children, the emotional excitement induced by these measures in many children renders their employment undesirable. The opposite method of treatment by muscular and neural stimulants, such as digitalis and strychnine, when uncombined with a preponderantly sedative agent, on the other hand, has been found to be of little use, and in the opinion of competent observers even harmful. "Digitalis," writes Dr. Sturges, "still, to quote my own opinion (though it is that of high authorities as well, notably of Dr. Stokes), is worse than useless in the acute cases we are now considering."* Rest of mind, so far as possible, or the avoidance and subjugation of emotional excitement in children, together with repose of body and the employment of calmative agencies general and local, is the treatment of carditis which the facts of experience have borne in upon the minds of physicians as most rational and most beneficial.

Among such calmative agencies there is none more powerful than opium, when there is no idiosyncratic intolerance of the drug, and when it is used with due care in children, in view of their special susceptibility to its action. There is reason to believe, indeed, that the fear of this special susceptibility has interfered with a sufficiently bold exhibition of it to attain the full benefit capable of accruing from its guarded but adequate use. The younger the

* 'Heart Inflammation in Children,' p. 75.

patient the greater is the care necessary in its use, but even young children when excited locally and generally by an actively irritative process can tolerate larger doses than in normal circumstances. Indeed, the physiological consequences of inflammatory reaction resemble in a measure the phenomena caused by agents known to be antagonistic to the action of opium. The quick pulse, the dilated pupil, the suffused skin, the uneasy sleep, or even wakefulness and delirium of the carditic child may be likened to the poisonous effects of the belladonna group of agencies. It is under these circumstances that the use of opium finds its most beneficial application. This fact is of course well known and has been mentioned by many writers, but rarely with the emphasis it deserves. Dr. Cheadle, however, relates a case in point.* "A boy of twelve, with mitral disease, adherent pericardium, and resultant engorgement of both lungs, had frequent pulse and palpitation with harassing incessant cough, which became so much aggravated on lying down that he could not sleep. He began to lose ground rapidly, chiefly from want of rest. He was then given ten drops of nepenthe every four hours. Rapid improvement followed, the cough gradually ceased, the pulse-rate fell from 120 to 90, good sleep was obtained and appetite returned. It is worthy of note how the opium in this case acted as a tonic; instead of impairing appetite it increased it by giving ease and sleep. This patient went out convalescent to return in a few months in almost exactly the same condition, sleepless from incessant cough due to congestion of the bases of the lungs. The same treatment proved efficacious a second time and he went out convalescent." Dr. Cheadle expresses a preference for the use of opium by the mouth, rather than hypodermically, regarding the latter method from its rapidity of action as not free from danger. As I shall presently relate, however, small doses of morphia given hypodermically may be well borne even by a young subject, provided the

* 'Lectures on the Practice of Medicine,' Smith, Elder and Co., London, 1900, p. 34.

physiological antagonistic conditions be well marked. Gibson also * speaks with approval of the hypodermic use of morphia, but his remarks are general and appear to apply rather to the adult than the child. Ashby † relates that in heart disease in children: "small doses of opium are usually required and are of the greatest use in relieving pain and quieting the heart's action. Half to two grains of Dover's powder may be given at night and repeated once or twice in twenty-four hours according to circumstances." Barié ‡ extols the value of hypodermic injections of morphia in pericarditis associated with pain, but prefers not to use it when there is evidence of myocardial weakness. On the general use of this ancient remedy in carditis and pericarditis no more need, however, be said. It is to encourage a more general and bolder use of it in the carditis of children that I have brought this matter before the Society.

With this view I relate the following case, although it had a fatal issue after a protracted illness, because it illustrates the tolerance of even a child for considerable doses of morphia when the physiological phenomena of general and local excitement are well marked, because also the general comfort of the patient was notably increased by such dosage, and finally because the post-mortem examination of the body revealed the anatomical conditions which underlay the phenomena manifested.

Louisa V—, aged 5 years and 7 months, was admitted under my care into the Children's Hospital, Paddington Green, on February the 18th, 1903. She was stated to have been ill for two weeks and complained of pain in the epigastrium and precordia. She was also stated to have been very feverish and had pains in the elbows and knees. Her appetite, however, was good, and the bowels regular, the urine was free from albumen, and there was no swelling of the feet or other sign of anasarca. She was apparently a highly neurotic, peevish and restless child, apart from

* 'Diseases of the Heart and Aorta,' p. 278.

† 'Diseases of Children,' 4th edition, 1899, p. 416.

‡ 'Traité Pratique des Mal. de Cœur,' Paris, 1900, p. 213.

her illness. No history of previous illness could be obtained, except that she had had measles. She was one of five children, the other four of whom were stated to be healthy. No history of rheumatism could be elicited, but the child's maternal grandmother was alleged to have had a "bad heart." The tenement occupied by the family was near the neighbouring canal and the house was said to be damp.

The patient after admission maintained the orthopnœic position, and had manifest difficulty of breathing. The area of cardiac dulness was increased, measuring four inches transversely from the right edge of the sternum to beyond the apex-beat in the fifth left intercostal space, and two and a half inches vertically from the lower edge of the second left rib cartilage to the fifth intercostal space. There was visible pulsation all over the precordia and the epigastrium. There was a loud systolic bruit at the apex, completely obscuring the first sound of the heart, conducted outwards to the left, and audible in the back. There was likewise a diastolic bruit at the apex. There was a systolic bruit in the aortic area, and both second sounds were without bruit. In the tricuspid area systolic and diastolic bruits were audible, and there was marked reflux into the internal jugular veins. The area of cardiac dulness did not vary in size, and it was assumed, therefore, that there was no pericardial effusion, nor was there any pericardial friction. The pulse-rate varied from 108 to 120 to the minute, and its volume was good. The lungs anteriorly were free from evidence of engorgement although entry of air was not forcible. Both lung bases posteriorly had a flat percussion note, with crepitation and defective respiration. The respiratory-rate was high, varying from 58 to 72 to the minute. The temperature was 99·2° F. It would be tedious to relate at length the daily changes in the condition of the patient; I shall therefore only indicate salient features in its progress, and reserve for fuller treatment the period during which the patient was more particularly under the influence of morphia, as that is the proper burden of our present theme. The range of temperature throughout the

case while under observation varied from a little above normal (99° to 100° F.) to about a degree below normal (96·8° to 97·8° F.). The pulse-rate was highest during the first month after admission, ranging from 130 to 150 to the minute, after which it fell to an average of 120 to the minute for the morning, sometimes slightly more, sometimes less, and was slightly higher as a rule in the evening than in the morning. The rate of respiration was likewise as a rule higher in the evening than in the morning and reached its highest general range about a fortnight after admission (March the 7th, 1903) and continued high (52, 60, 64, 68, and 72 to the minute) until the end of March. Small doses of opium were first added to the medication on the 4th of March, namely, two minims of tincture of opium every six hours. This had no appreciable effect. An additional minim per dose was added on March the 16th with as little result. Four minims of nepenthe were given every four hours after the 30th of March, apparently with somewhat more benefit, the pulse-rate and respiration becoming quieter and the child more comfortable. On the 5th of April the dose of nepenthe was raised to six minims with apparent benefit. This was, however, but a timid feeling of one's way as regards the effect of opium, and was merely an adjunct to other attempted means of relief. Leeches were from time to time applied over the liver, a procedure which the child keenly resented, and ultimately cried bitterly if these were mentioned in conversation within her hearing. Ice was applied to the precordia, as much against her will as the leeches were to her right hypochondrium. Both procedures added much to her excitement and unrest and had to be abandoned. The patient was evidently becoming progressively worse. She slept little, constantly cried out as if in pain, and continuously taxed the efforts of the nurses, who assiduously attended her day and night. The legs and a portion of the trunk and the face became œdematous, the liver enlarged and pulsated, the bases of the lungs became more engorged, and the cardiac area was increased, but without evidence of fluid. The legs were

satisfactorily drained by Southey's tubes and the general anasarca of the trunk and face greatly diminished.

On the 9th of April, finding the chief deteriorating factors were the continuous restlessness and sleeplessness of the patient and the progressive dilatation of the heart, I prescribed hypodermic injections of $\frac{1}{200}$th of a grain of digitalin, $\frac{1}{100}$th of a grain of strychnine and $\frac{1}{16}$th of a grain of morphia to be given, with careful observation as to general effects, every four hours. During the first twenty-four hours four doses were given, amounting altogether to $\frac{1}{4}$th of a grain of morphia. The hypodermic injections were accurately measured and freshly prepared by the dispenser to the hospital. The morning pulse-rate on the 9th of April was 132 to the minute, the evening 164 to the minute. The respiratory-rate in the morning was 62 to the minute, in the evening 68 to the minute. The temperature in the morning was 97° F., and in the evening 98·2° F. The next day the precise dosage of morphia is not noted on the chart, but it was prescribed to be used as conditions necessitated. She is stated on the 12th of April to have had a quiet night. The pulse-rate was noted as 124 to the minute and the respiration-rate as 24 to the minute. The pupils were beginning to react to opium. During the next twenty-four hours, however, the child must have become more restless, for the hypodermic injection of morphia had to be administered every four hours, amounting in all to about half a grain in twenty-four hours. On the 13th of April the patient had in all $\frac{1}{8}$th of a grain and slept only three hours during the night, but slept well after daybreak. The respiratory-rate is noted as 32 to the minute and the pulse as 112 to the minute. On the 17th of April, although the patient had each day (with the exception of the 16th, when she had $\frac{1}{16}$th of a grain) $\frac{1}{8}$th of a grain of morphia hypodermically, the pupils are recorded as not contracted. The general condition of the patient continued one of comparative comfort until the 25th of April, although her local signs showed little amelioration. One of my colleagues in passing through the ward remarked to the house-

physician, "That child looks better." On that date, however, an increase of morphia to $\frac{1}{5}$th of a grain in twenty-four hours was necessitated, and next day the child needed $\frac{1}{4}$th of a grain, and the same amount for two days afterwards. On the 28th of April the record is that the child had had a fair night, having slept from four to five hours in snatches; and the pupils are stated to have been "rather smaller." The pulse-rate was from 138 to 144 to the minute, and the respiratory-rate from 32 to 36 per minute, while the temperature was 97·6° F. to 98·2° F. There was œdema of the left lung apex anteriorly, but not much congestion of the bases; the liver edge was half an inch below the umbilicus, the pulse was regular and of good volume, and the jugular distension was less marked. As the patient had been sick three times on the 28th, the strychnine and digitalis were omitted. For the next three days only $\frac{1}{16}$th of a grain of morphia was given, the digitalis and strychnine being resumed on the 30th of April as the cardiac failure was greater. The patient died at 4.15 a.m. on the 1st of May. For careful notes of the case I am indebted to Dr. Carey, recently house-physician to the Children's Hospital, Paddington Green. On post-mortem examination, the pericardium was found to be adherent to the costal pleura from the fourth to the sixth ribs on the left side, and to both the lungs. The parietal and visceral layers of the pericardium were universally adherent obliterating the sac. The heart likewise adhered to the diaphragm. The transverse diameter of the heart was much increased and the surface veins of the organ were engorged. The total weight of the heart including the contained blood and attached vessels was thirteen ounces. All the cavities of the heart contained blood-clot. All the valves were normal, except the mitral on its auricular surface where there was a fringe of granulations. The auriculo-ventricular orifices admitted two fingers. The right auricle was dilated. The right ventricle was not dilated and in fact appeared to be rather smaller than usual. The left auricle was not markedly dilated. The left

ventricle was both dilated and hypertrophied. There was atheroma at the orifices of the coronary arteries, but the arteries themselves were healthy. Both lungs were congested chiefly at the bases. There were no infarcts and no evidence of tuberculosis here or elsewhere. The capsules of the kidneys stripped easily, the surface veins were engorged, and the cortex was somewhat increased in depth, and although the urinary output was small and difficult to collect during life, the urine was only rarely found to contain a little albumen from circulatory stasis. On microscopic examination, the pericardium was thickened and of organised structure. Vessels in it were found affected by endarteritis and periarteritis with changes in the muscle nuclei. Nerve-trunks were seen involved in the pericardial inflammation. The interstitial texture of the myocardium was thickened and increased in quantity and the nuclei were increased in number from proliferation, but there was no evidence of excessive connective-tissue proliferation of recent date. The muscle-fibres had in places well-marked transverse striæ, but this was not well marked in the majority of the fibres.

Remarks.—There are many questions raised by the consideration of this subject which the scope of this paper and the limits of time preclude my discussing: such as the comparatively recent or more chronic nature of the inflammatory process necessary to bring about a symphysis of the pericardium such as this case revealed; the cause of death from heart disease in children the muscular fibre of whose hearts is well preserved—a point to which Dr. Sturges draws special attention;—and the influence in preserving or prolonging life of various therapeutic measures. Although with Dr. Lees we may have "given up the idea of curing pericarditis," we may, nevertheless, agree with Dr. Sturges, who writes on the whole rather pessimistically of the therapeutics of carditis in children when he remarks, "The easing of pain by local appliances, the procuring of sleep therewith, the timely withdrawal of blood to relieve venous congestion and

cardiac dyspnœa,—all these measures are truly life-saving; but the course of inflammation is little, if at all, affected by such means," (op. cit., p. 74). I have already indicated my belief, that the mechanisms by which some of these measures save life is chiefly by their action upon the cardiac nervous system, also that they are avowedly directed against effects, not a primary cause, and that although they may not directly affect the "course of inflammation"—a point which can scarcely, in my opinion, be regarded as indisputable,—they may help to maintain life until the conflagration has burned itself out, or the protective processes of the body have eliminated the intruding cause of disease. Among such useful measures, I feel disposed to give a prominent place to opium, and it is with a view of emphasising the capacity of children for tolerating considerable doses of the drug, under suitable circumstances, that I have ventured to draw the attention of the Society afresh to the subject of its employment in the carditis of the young.

(*Read at the Provincial Meeting, Brighton, June the 20th*, 1903.)

68. SOME CASES OF DILATATION OF THE BRONCHIAL TUBES AFTER MEASLES.

By THEODORE FISHER, M.D.

In the post-mortem room from time to time one meets with dilatation of the bronchial tubes in children. The dilatation may be widely distributed through both lungs, or may affect only one lung, or perhaps only one lobe of a lung, which may be the upper or the lower lobe. In other cases a lobe in each lung may show marked dilatation of the bronchial tubes, in which case it is generally both lower lobes. Occasionally, when the dilatation of the bronchial tubes is limited to one lung or one lobe of a lung, a definite

local cause in the form of some obstruction to the bronchus or one of its main divisions may be found. For example, an enlarged caseous bronchial gland may in rare instances give rise to such obstruction. In other cases, again, a history of a foreign body having been at one time present may be obtained. A history of this character, however, is not common, but it is possible that in some cases in which no history suggesting the presence of a foreign body can be obtained, such a body may have been present. The fact that nothing of that nature is found post-mortem in the bronchus does not prove that such a body was not at one time present. For example, in one case recorded by Mr. C. H. Morton, which I saw clinically and in the post-mortem room, where a history of a small steel body having entered the air-passages was definite, and the child's illness dated from that event, at the autopsy no trace of the foreign body nor other cause of obstruction could be found in the bronchus of the affected lung, although the dilatation of the bronchial tubes was general and well marked throughout the lung. The steel body must either have rusted away or have been expectorated without attracting attention. In other cases, where sweets or portions of nuts enter the air-passages, solution or disintegration may possibly occur. In connection with the mention of sweets an illustration may be given of the difficulty of obtaining a reliable history, even from intelligent parents, of the entrance of a foreign body into the air-passages. In one little girl brought to the Bristol Hospital for Sick Children for chronic lung trouble, I found what I considered to be obvious evidence of dilatation of the bronchial tubes in the lower lobe of the left lung, yet careful questioning of the mother failed to elicit any facts suggesting the entrance of a foreign body into the air-passages. On the next visit, however, the mother apologised for having forgotten that about nine months previously when the child was sucking an "acid-drop" the sweet had "gone the wrong way." Violent fits of coughing followed which left the child very exhausted. A doctor was called in four days later who

found the child feverish, and it was considered necessary to keep her away from school for a week. The troublesome cough from which the girl suffered had dated from that time. While some cases of dilatation of the bronchial tubes in children are due to pressure upon a bronchus or one of its divisions by a caseous gland, and others to blockage of the bronchus or of one of its divisions by a foreign body, some cases undoubtedly occur as a sequel of broncho-pneumonia. Curiously enough, the last four instances of dilatation of the bronchial tubes of this character met with by myself have apparently been occasioned by broncho-pneumonia occurring as a complication of measles. In all the cases the children were said to have been free from any indication of disease in the chest until they were attacked by measles. Within a few weeks or months of the onset of measles death occurred, and dilatation of the bronchial tubes in varying degree was found. The first, and also the least marked case, because death took place earlier than in the other cases, was met with at the Bristol Hospital for Sick Children, when I was taking the work of the senior physician, Dr. Elliott.

Case 1.—A girl, aged 3 years, was brought to the hospital with broncho-pneumonia four weeks after an attack of measles and was admitted. The child was obviously very ill; the pulse-rate 170 to the minute, the respirations 72 to the minute, and the temperature 100° F. Numerous sharp râles were heard over both sides of the chest, but especially over the right side; and we were doubtful whether we had a case of broncho-pneumonia or of acute tuberculosis. Nine days later the child died. At the autopsy the greater part of the right upper lobe was found to be solid, and the bronchial tubes everywhere were slightly but definitely dilated, giving a sieve-like appearance to the lobe. Patchy consolidation was present in the middle and lower lobes, and the affected areas showed similar dilatation of the bronchial tubes. In the left lower lobe were very small scattered areas of broncho-pneumonia,

but no obvious dilatation of the bronchial tube was present.

In the next case the dilatation of the bronchial tubes was very much more marked.

Case 2.—A boy, aged 1 year and 8 months, was brought to the out-patient department of the Bristol Children's Hospital on January the 26th, 1903. The child had the pale, puffy face not uncommonly seen in children with chronic lung trouble. The fingers were slightly clubbed and the breath offensive. The physical signs of the lungs showed a somewhat dull note over the whole of both lower lobes, and marked bronchial breathing was present. On inquiring into the history it was found that the child had suffered from measles when eight months of age and had been affected with cough since that time. This fact, together with the aspect of the child, clubbed fingers, and offensive breath, led me fairly confidently to enter in my note-book, "Dilated bronchial tubes after measles," with recent broncho-pneumonia. The child died four days later and the autopsy was performed by Dr. Bertram Rogers. Both lower lobes were found to be adherent to the chest wall and somewhat contracted. On section of the lungs the bronchial tubes were seen to be everywhere greatly dilated and their walls thickened. Some of the cavities were large enough to hold a medium-sized pea. The upper lobes showed no dilatation of the bronchial tubes, but small patches of broncho-pneumonia were present. On microscopical examination, the lower lobes showed no general fibrosis. There was considerable increase of fibrous tissue with destruction of alveoli immediately around the dilated bronchial tubes, but for the most part the intervening lung substance was unaffected except that recent broncho-pneumonia was present. A very noteworthy feature of this case, which I have seen in other cases of dilated bronchial tubes in children, was great thickening of the blood-vessels, due apparently to chronic inflammation of the perivascular lymphatic spaces.

The next two cases were met with in the post-mortem room of the Bristol Royal Infirmary.

Case 3.—A boy, aged 1 year, was admitted into the Bristol Royal Infirmary on November the 21st, 1902, under the care of Dr. Shaw. He had suffered from an attack of measles four months before, and since that time a cough had always been present. He had been worse during the

Fig. 29.—Photograph of a section of the right lung from Case 3.

week before admission. Wide-spread bronchophony was present over the right side of the chest but loudest over the upper lobe, where cavernous sounds were also present. The temperature oscillated between 100° F. and 102° to 103° F. The child died fifteen days after admission. At the autopsy marked dilatation of the bronchial tubes was present in both lungs, but chiefly in the upper lobe of the right lung, and to a less extent in the lower lobe of the left lung.

The cut surfaces of the lungs were dotted over with cavities of various sizes, some of which were large enough to hold a good-sized pea, even in the right upper lobe close under the pleural surface. Some patches of consolidation were also present. In the following case the dilatation of the bronchial tubes was of the same character, but much less extensive.

Case 4.—A boy, aged 17 months, was admitted into the Bristol Royal Infirmary, under the care of Dr. Waldo, with raised temperature, rapid respiration and pulse, and died a few hours later. He was said to have been ill since an attack of measles three months before. At the autopsy the left lower lobe was found to be adherent to the chest wall and the pleura was greatly thickened where the lung was adherent to the diaphragm, measuring at some places fully a quarter of an inch in thickness. The bronchial tubes throughout the lobe were markedly dilated and their walls thickened. In some places thickening of the smaller blood-vessels could also be seen. Amongst the dilated tubes were patches of recent consolidation. The rest of the lung and the whole of the right lung were quite healthy and free from dilatation of the bronchial tubes. Earlier in the year 1902 I had met with a case in the post-mortem room where dilatation of bronchial tubes in the lower lobes was so well-marked that I made a sketch of the condition found. On looking up the clinical notes I found it merely stated that the boy, who was aged 14 years, had been ill for nine years. I therefore paid a visit to the parents, who gave me the following interesting history. After I had mentioned in the form of a question that the notes stated that the boy had been ill for nine years, I received as a reply, "Yes, and he had measles five times." Further questioning seemed to show this to be true. The boy had measles when three years old; and again when four, at a time measles was present in the house of friends; there was a third attack at the age of seven, when one sister was suffering from an attack of measles; and again when aged nine, two other sisters having this time brought the disease home from

school. Lastly, he suffered for a fifth time when aged thirteen during an epidemic of measles in Bristol.

Whether the apparently definite association of the dilatation of the bronchial tubes with measles in these cases has been merely a coincidence, or whether dilatation of the bronchial tubes is more likely to follow the broncho-pneumonia which often complicated measles, is difficult to say. I have seen dilatation of the bronchial tubes to slighter extent follow diphtheria, and also in two cases where a history only of repeated attacks of bronchitis could be obtained.

Reference to the papers of Carr* and Sharkey† do not seem to strongly support the idea that measles plays a very important part in the production of dilatation of the bronchial tubes in children. A history of this disease is, however, mentioned in two of Carr's cases and one of Sharkey's. Yet, although one cannot definitely say that the broncho-pneumonia of measles is a more potent factor in causing dilatation of the bronchial tubes than bronchitis, or broncho-pneumonia occurring alone, or as a complication of other diseases, the curious fact, however, that in four successive cases that have come under my notice the disease of the lungs appears to have dated from an attack of measles, and that in a fifth several attacks of measles have been present, seems to be worthy of record.

(*Read at the Provincial Meeting, Brighton, June the 20th*, 1903.)

69. NOTES ON AN UNUSUAL COURSE OF A CASE OF CEREBELLAR TUMOUR.

By ELDON PRATT, M.D.

The patient, a female child aged 14 years, was admitted into the Cardiff Infirmary on October the 21st, 1895. Her

* 'Practitioner,' February, 1896, p. 87.

† 'St. Thomas's Hosp. Reports,' vol xxii, p. 33.

mother gave a six months' history of vomiting, especially severe for two months at the beginning of the illness, staggering gait, giddiness, and latterly loss of sight. Although only a Board School child, she showed a marked musical talent. Besides doing a good deal of study, she gave music lessons amongst the poorer classes, but of late had had to give up all work.

On admission she seemed to be somewhat mentally blurred. Frequent complaints were made of headache, and there was loss of sight, double optic neuritis being very marked. There was deafness of the left ear, but no history of aural discharge; the deafness, however, was quite recent. Other senses, carefully tested, were found to be normal. There was a good deal of retraction of the head and pain on tapping the spinal column all the way down. Her gait was very staggering. The knee-jerks were absent and there was no paralysis. She was unable to hold her water and she was very constipated. She got progressively worse, headache being extremely severe. She seemed to be rapidly dying, her colour was very bad, and she was apparently sinking into a state of coma; in fact, the case was regarded as hopeless. As a last resource, just three weeks after her admission into the hospital, a seton of the old-fashioned, broad ribbon kind was introduced through the nape of the neck, and from that time the change in her condition was really remarkable. Gradually the headache, the vomiting, the retraction of the head, and optic neuritis, and, later, the obstinate constipation and giddiness, all entirely disappeared. Having been in hospital just over four months, she was discharged on February the 19th, 1896, apparently quite recovered except for total blindness, atrophy of both optic discs having resulted, and deafness in the left ear, and loss of the sense of smell, which, although normal on admission, was found to have disappeared when tested during convalescence.

After leaving the hospital she enjoyed a few months' holiday, and then resumed her studies, eventually entering the Normal College for the Blind at Norwood. Here she so

far distinguished herself as to obtain an exhibition at the Royal Academy of Music, tenable for two years. Her success, however, was doomed to be short-lived, for within five weeks of her leaving Norwood she was again in hospital with a recurrence of all the old symptoms. She was readmitted on September the 2nd, 1897. The headache was severe and nearly always occipital; the vomiting, constipation, giddiness, and staggering all recurred. A new symptom was complained of, namely, pain in the right shoulder shooting through to the breast, and she said that the pain once went down the right arm so that for a short time after she was unable to lift that limb. Retraction of the head recurred within three days of her readmission, and the seton was again introduced into the nape of the neck. The headache, however, increased in severity. She became gradually worse, and on October the 1st she fell into a state of drowsiness, the colour of her features being purplish, and she suddenly expired that same evening.

At the post-mortem examination the head only was inspected. The dura mater did not present the normal uniform shiny appearance, but was mottled with patches of probable old inflammation; there was certainly no recent meningitis. All the ventricles of the brain were immensely dilated. In the left hemisphere of the cerebellum was a tumour about the size of a walnut, superficial in position, discrete, rather hard and yellowish in colour, and on section its centre was somewhat caseous. It was probably a tuberculous mass. In the same hemisphere was also a cyst, larger in size than the tumour, and having no communication with other parts. The tissues of the left hemisphere were much thinned out; other parts of the brain than those mentioned were natural.

Here, then, briefly, are the facts of a case of cerebellar trouble extending over a period of two and a half years. The chief point of interest seems to me to be the almost complete recovery from the first attack, and I venture to think that the seton was at least, for the most part, instrumental in such recovery, for the effect, when the patient

was apparently near her end, was too marked to be otherwise. I think it probable that in that first attack she had also some meningitis, which would perhaps account for the loss of the sense of smell, and for the loss of hearing in the left ear. Remarkable, too, is it that she should have left the hospital with her intellect so unclouded as to be able a few months later to obtain by competition a scholarship in music. Possibly, the extra strain thus involved hastened her end by renewing to a severer degree all the old symptoms of the first attack.

(*Read at the Provincial Meeting, Brighton, June the 20th*, 1903.)

70. ON A COMMON APPEARANCE OF THE OPTIC PAPILLA LIKELY TO BE MISTAKEN FOR OPTIC PAPILLITIS.

By SYDNEY STEPHENSON, C.M.

When a physician uses his ophthalmoscope he generally searches for a few well-defined fundus changes, of which optic neuritis is undoubtedly the chief. It is therefore of the first importance that he should know how to avoid any pitfalls that may lie in the way of the diagnosis of the latter condition.

Amongst children there is an appearance of the optic papilla that is sometimes spoken of as the "*hypermetropic disc*" or "*pseudo-neuritis.*" It constitutes in many cases a very serious source of fallacy in the diagnosis of papillitis. Its existence, of course, is familiar to every ophthalmic surgeon, but I feel sure, from my inquiries, that many physicians either ignore its existence altogether or else attach too little importance to it. The following figures, nevertheless, go to show that "pseudo-neuritis" is extremely common amongst children generally. I have

FIG. 1.—Left eye, erect image.

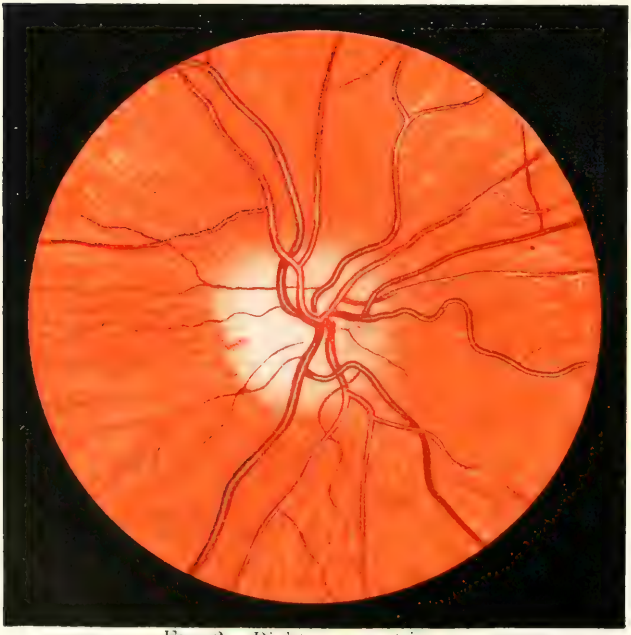


FIG. 2.—Right eye, erect image.

Cases of "Pseudo-neuritis" of the optic papilla. Illustrating **Mr. SYDNEY STEPHENSON's** paper, "On a common appearance of the optic papilla likely to be mistaken for optic papillitis."

recently examined 114 children, belonging to both sexes, whose ages ranged from two to sixteen years, and who enjoyed good health. The eyes were primarily examined for the purpose of estimating the refraction, but in doing this I paid particular attention to the existence of the condition in question. I found that of the total number of the children as many as 26—that is, 22·80 per cent.*—presented a greater or less degree of "pseudo-neuritis." Speaking broadly, then, about one child in every four and a half of those examined was affected with "pseudo-neuritis."

The ophthalmoscopic appearances of "pseudo-neuritis" in many instances, as I have said before, simulate those of true optic neuritis (*vide* Plate IV, fig. 1). So much is this the case that it may be quite impossible to differentiate between the two conditions upon a first examination of the eyes. Indeed, so striking is sometimes the likeness that I am convinced in my own mind that of many cases diagnosed as "optic neuritis" by physicians some must really have belonged to the spurious group.

The slighter grades of pseudo-neuritis present a blurring or haziness of the nasal edge of the disc, closely resembling a commencing optic neuritis (*vide* Plate IV, fig. 2). Associated with this two other appearances are commonly met with, namely: (1), more or less glistening connective tissue alongside the central retinal vessels on the papilla; and (2), some tortuosity of the retinal arteries and veins on the fundus generally. More pronounced cases of "pseudo-neuritis" show a distinct woolliness of the nasal edge of the disc, which is greyer than normal. In more advanced grades still, the entire margin of the optic disc becomes ill-defined, diffuse, and occasionally striated, and the condition is difficult to distinguish from a developed descending neuritis. In the most pronounced grade of all, it becomes difficult or im-

* It is rather a singular coincidence that Dr. Herbert Carpenter Bristowe ('Ophthalmic Review,' vol. x (1891), p. 321) found the condition to be present in 29 amongst 125 patients, *i. e.* in 23·2 per cent., or almost the same proportion to that given above. Some of his best marked cases were in persons over 30 years of age.

possible to say where the disc ends and the fundus begins, so hyperæmic and indistinct does the former appear. It will thus be seen that while the slighter cases of "pseudo-neuritis" may be mistaken for a commencing optic neuritis, the more marked cases may be confused with a well-developed optic neuritis. In point of fact, the mistake is not only excusable, but, under some circumstances, almost impossible to avoid. Finally, it should be added that the change, although invariably bilateral, is not necessarily equal in degree as regards the two eyes.

A situation of some uncertainty is thus created. How can we distinguish the two conditions, neuritis and "pseudo-neuritis," from one another? The main points in the differential diagnosis appear to me to be the following: 1st. "Pseudo-neuritis" is a congenital condition, neither receding nor advancing, no matter how long a case remains under observation. True neuritis, on the other hand, begins, develops, reaches its height, and is then replaced by atrophy of the disc or (in very rare cases) by *restituo ad integrum*. Therefore, if we find that a blurred state of the optic disc supervenes while a patient is under observation, or if it becomes more pronounced under those conditions, we may safely conclude that we are dealing with a true optic neuritis.

2nd. "Pseudo-neuritis" of itself causes no disturbance of vision, although some degree of so-called congenital amblyopia not infrequently complicates the case. But, as Hughlings Jackson long ago insisted, even advanced optic neuritis may exist without compromising sight seriously. Hence, the mere visual acuity can scarcely be depended upon either way for purposes of diagnosis. At the same time, if we come across what appears to be a well-marked optic neuritis co-existing with normal sight, the presumption will favour "pseudo-neuritis" rather than true neuritis. I have never met in "pseudo-neuritis" with those oft-recurring and transitory obscurations of sight that are so marked a feature of some cases of optic neuritis.

3rd. The visual field in optic neuritis, as everybody

knows, usually shows some peripheral contraction, particularly for colours, or it may be of the hemiopic type. In "pseudo-neuritis," on the contrary, such changes are never found. It is, however, unfortunate that the tender age of many patients precludes an appeal to this valuable diagnostic point. Still, the visual field may be mapped out with fair accuracy in most children over seven or eight years of age.

4th. The disc, in true neuritis, is swollen in all its dimensions, a change that, if at all well marked, reveals itself to ophthalmoscopic examination by a difference between the refraction at the disc and in the yellow spot region of the fundus. I have never been able to satisfy myself that such a difference existed in "pseudo-neuritis," even although to other tests, notably the appearance of the vessels, the disc appeared to be distinctly swollen. Accordingly, I am inclined to lay great stress upon this as a diagnostic point between the two conditions.

5th. Hæmorrhages and white spots or splashes are never present in the neighbouring retina in "pseudo-neuritis," whereas they are often present in true neuritis. Dilated and motionless pupils, again, although they may accompany the latter condition, never do the former. But in this connection it should be remembered that the dilatation of a pupil is connected more closely with defective sight than with the degree of optic neuritis.

The exact etiology of "pseudo-neuritis" is not known, although my investigations render it certain that it is a congenital condition. Some authors seem to regard it as the expression of an actual hyperæmia or congestion of the optic disc, due to eye-strain produced by hypermetropia or hypermetropic astigmatism. Hence the name the "hypermetropic disc." With this view I cannot agree, and for two cogent reasons. First, if "pseudo-neuritis" is caused by hypermetropia, it should be relatively more frequent and certainly more pronounced in the higher than in the lower grades of that anomaly. The figures at my disposal, however, prove that such is not the case. Secondly, it should

undergo an improvement or even disappear altogether when eye-strain was relieved by suitable glasses, but I have often satisfied myself that such is not a fact. Besides, "pseudo-neuritis" may occur in association, not with hypermetropia, but with myopia or myopic astigmatism. If it is more frequent in hypermetropia it is simply because in children that condition is at least five times as common as the reverse condition, myopia. Indeed, I may say that my investigations justify me in claiming that in reality pseudo-neuritis is relatively as common in the one condition as in the other. The foregoing considerations lead me to renounce the name of the "hypermetropic disc" unconditionally.

In conclusion, I may be permitted to say that I look upon "pseudo-neuritis" as a congenital non-differentiation of the optic disc, a view supported by the frequent existence of shreds of connective tissue and also by the state of the retinal vessels.

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